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DISSECTING ANEURYSMS OF THE THORACIC AND ABDOMINAL AORTA

REPORT OF SIX CASES, WITH A DISCUSSION OF ROENTGENOLOGIC FINDINGS AND PATHOLOGIC CHANGES

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UNTIL the last decade, the diagnosis of dissecting aneurysm was rarely made during life. Now, as the result of many thorough studies of the disease processes and their physical manifestations, sufficient facts have been accumulated to allow the diagnosis to be made with relative frequency. This paper presents some of the facts and conclusions derived from a review of the literature and study of the material available at the State University of Iowa, with emphasis on correlation of pathologic and roentgenologic aspects. Six cases with roentgenologic findings are briefly described.

In dissecting aneurysm of the aorta, blood dissects through a tear or split in the coats to produce an intramural hematoma. It differs from other aneurysms in that the lumen is usually not enlarged by the dissection, but remains constant in size or is made smaller by the hematoma.

Most dissecting aneurysms occur in persons forty years of age or older. In the 300 cases analyzed by Shennan,[43] more than half (59 per cent) occurred after the age of fifty years, and approximately one-third after the age of sixty. They occur less commonly in children, although the most extensive aneurysm reported occurred in a fifteen year old boy.[12] Of Shennan's cases, 65 per cent occurred in men and 35 per cent in women, with little difference in sex incidence until after fifty years of age, when the percentage in men rose.

In the ten year period, 1939 to 1949, dissecting aneurysm of the aorta was found in 15 of 3,188 consecutive autopsies at the State University of Iowa, an incidence of 1 in 212. Holland and Bayley[21] have found an incidence of 1 in 381 autopsies, which is essentially in agreement with most reported figures.

This study indicates that there are in general two types of dissecting aneurysms of the aorta. The first, here referred to as the classical type, is associated with widespread necrosis of the media, often with extensive dissection of the coats. The second, or arteriosclerotic type, is charac-

terized by severe arteriosclerotic changes in the intima and media preceding dissection.

DISSECTING ANEURYSM OF THE CLASSICAL TYPE

The strength and elasticity of the normal wall of the aorta lies chiefly in the media, which consists of compact layers of smooth muscle fibers alternated with wavy bundles of elastic fibrils. Specific degenerative changes in the media lead to the initiation of dissection in the classical type of aneurysm. These changes have been classified into five types by Sailer,[41] and involve the collagenous and elastic tissue, muscle, and ground substance of the media in varying degrees. Cystic changes in the media, first described by Erdheim,[7] occur mostly in the ascending portion of the aortic arch. Rottino[40] examined 210 aortas from routine autopsies for evidences of cystic changes, and was able to find them in only 7.

In 6 cases of classical dissecting aneurysm, 3 of which are not presented here because of lack of roentgen studies, the changes seem to agree essentially with Types 1, 3 and 4 (Sailer) (Fig. 1 and 2). Extensive hyaline changes in the interlamellar connective tissues (Type 2) do occur in some of the aneurysms of the older age groups, but these are believed to stem from arteriosclerosis. No instance of Type 5 was found in this series. A most striking finding, however, was the remarkable extent of the necrosis in 2 cases aged twenty-four and twenty-six. This necrosis involved both elastic and muscle elements throughout the entire media of some segments, and was apparently so acute that there was little evidence of repair. Such extensive necrosis results in great weakness of the aortic wall, so that the layers may be separated by rubbing them between the fingers.

FIG. 1. Medial necrosis with mucoid degeneration. High power magnification, illustrating extensive destruction of elastic lamellas, and mucoid degeneration of interlamellar smooth muscle. Note the absence of inflammatory response.

FIG. 2. Medial necrosis, nonexudative degeneration of smooth muscle. High power magnification. The elastic lamellas with their characteristic undulations are intact. Note the band of interlamellar necrosis, with total absence of nuclei. There is no inflammatory response.

The etiology of such degeneration of the media is obscure. Atherosclerosis, medial necrosis and dissecting aneurysm have been produced experimentally in rabbits by repeated injections of epinephrine.[7,27] Arsphenamine, nicotine, alcohol, cholesterol, and diphtheria toxin have also been

suggested as agents toxic to the media. A specific toxic agent in syphilis has been suggested by Gsell,[16] but its existence has never been proved. Dissecting aneurysm has occurred during the last trimester of pregnancy in numerous cases.[13,28,33] The lesions of rheumatic fever were believed to be responsible in at least one instance.[15] Congenital medial deficiency and medial thinning, observed in connection with developmental anomalies such as arachnodactyly and coarctation of the aorta, may be responsible for some congenital and dissecting aneurysms of the aorta.[1,4,17,46] Recently, some very important evidence has been uncovered, by Ponseti and Baird,[36] who in studying the etiology of scoliosis, fed a diet of 50 per cent sweet peas to growing rats. He was able to produce scoliosis in rats fed with this diet, but also found that 30 per cent of his animals developed dissecting aneurysm of the type described here as classical.

There is considerable difference of opinion regarding the relationship of syphilis to dissecting aneurysm. Shennan did not find syphilis a prominent factor in the etiology of dissecting aneurysm; and Sailer,[41] in reviewing the literature on dissecting aneurysm associated with syphilis, concluded that syphilis hinders the development of dissecting aneurysm through scarring of the media. Weiss[45] concurs in this opinion. Others believe that syphilitic aortitis neither prevents nor predisposes to the development of this condition.[37] The intimal scarring, thickening, and cicatrization which accompany syphilitic aortitis frequently undergo atherosclerotic degeneration with calcification when the aortitis is scarred and inactive.[24] Such calcification is found in the typical distribution of syphilis.[22]

Location and Mechanism of Internal Rupture. In the classical type, the chief sites of internal rupture are the ascending aorta a few centimeters above the aortic valve and the descending aorta near the isthmus and ligamentum arteriosum.[43] These two points are the junctions of the relatively immobile portions of the aorta with the less firmly supported arch,[37,38] and interestingly enough, are also the sites frequently involved in traumatic rupture of the aorta.[11] The first few centimeters of the ascending aorta is enveloped in the superior reflection of the pericardium, and is in contact with the distended pulmonary aorta and right pulmonary artery, which immobilize this portion of the vessel. The thoracic aorta, beginning at the level of the fourth thoracic vertebra, is firmly fixed by the root of the left lung, the intercostal arteries, the spine, and the reflections of the left pleura.[43] Wilens,[46] in his investigation of the changes of senility in the aorta, has observed that the immobilized segments of the aortic wall are the first to lose their elasticity. This fact may indicate that the aorta in these areas is potentially weaker.

Loeffler[28] believes that the abrupt diastolic recoil following closure of the aortic valves causes longitudinal stretching of the ascending aorta. This added strain may in some measure account for the high incidence of rupture in this region. In both the ascending aorta and the region of the isthmus, tears of the intima and media are most often transverse. In 1 of Shennan's cases the tear passed spirally around the

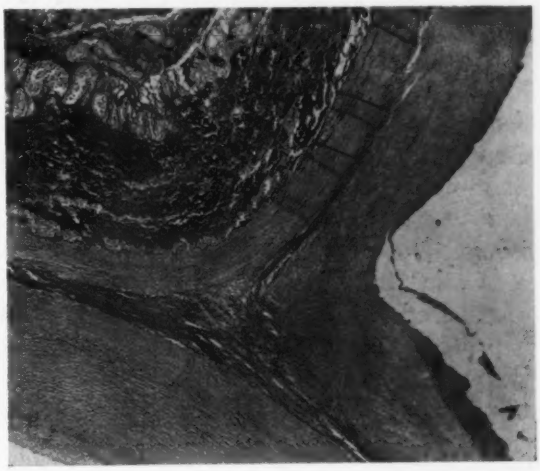

Fig. 3. The dissection. The layers are separated at the junction of the middle and outer thirds of the media, and "healing" has occurred. Note the atherosclerosis of the new intima.

circumference of the ascending aorta one and one-half times. Longitudinal, oblique, and serrated or angular tears may also occur.[43]

Dissection occurs most frequently at the junction of the middle and outer thirds of the media (Fig. 3), progresses proximally, distally, or in both directions, and involves all or a portion of the circumference of the aorta. If the descending aorta is completely encircled, the intercostal arteries may be divided within the aortic wall, and may receive their blood supply from the dissecting hematoma. Dissection is also prone to extend into the walls of the greater branches of both the thoracic and abdominal aorta. The extent of the dissection is extremely variable. In the most extensive case reported it extended into the posterior tibial artery of the left leg.[12] The dissection in the classical type is usually more extensive than that found in the arteriosclerotic type, but it may be limited because of external rupture and death.

Shennan, Sailer, and others, in studying cases of dissecting aneurysm due to idiopathic medionecrosis, have found that rupture rarely occurs through atheromatous plaques and ulcers in the intima.

It is interesting to speculate which occurs first, the rupture or the dissection. The belief most widely accepted is that the medial degeneration with cyst and cleft formation is responsible for intramural hemorrhage which expands, dissects, and secondarily ruptures into the aortic lumen. Reports of dissecting aneurysms in which no medial or intimal rents were discovered lend considerable support to this opinion. Bourne and Mills[3] have recently reported a a case in which multiple hematomas were discovered in the media without intimal rupture. Shennan believed, however, that the pressure from such minute vessels as the vasa vasorum is less likely to cause rupture of the media and intima than is the internal pressure of the aorta, and that internal rupture is a prerequisite to dissection. It is probable that either mechanism may, under favorable circumstance, effect the initial dissection.

Once dissection occurs, a sac is produced which bulges into the lumen of the aorta and tends to divert the blood stream into the intimal tear and the new intramural lumen. In smaller vessels, such behavior often results in total occlusion of the vascular lumen, but in the aorta, either the lumen is only partially occluded or secondary intimal tears allow the circulating blood to re-enter the main lumen. These secondary ruptures occur frequently at the level of the diaphragm or at the bifurcation of the abdominal aorta. In Case IV, the transient cyanosis of the lower abdomen and extremities leads one to suspect that there was momentary total occlusion of the descending aorta, with circulation re-established by re-entry. In Shennan's series of 218 aneurysms, 26 showed re-entrance ruptures. The percentage of re-entrance ruptures in "healed" aneurysms is somewhat higher. Jones and Langley[23] have further analyzed some of Shennan's figures, and have concluded that when re-entry did not take place, 96 per cent of the patients died in five weeks, whereas when the circulation was re-established, only 28 per cent died in the first five weeks. Re-entry occurs most frequently when an obstruction, such as the diaphragm or bifurcation of the abdominal aorta, is reached.

Death was due to external rupture of the aneurysm into the pericardial sac in 70 per cent of Shennan's cases.[43] Other sites of external rupture are into the pleurae, esophagus, mediastinum, pulmonary artery, right auricle, peritoneal cavity, and the retroperitoneal tissues. A small percentage of cases die of cardiac failure.[10] Approximately 10 per cent of dissecting aneurysms heal, and acquire secondary circulation through the channel of the dissection.[44] In a small number of these cases, endothelization of the lining of the new channel occurs (Fig. 3) and atheromatous changes with calcification may take place in this new intima.

DISSECTING ANEURYSMS OF ARTERIOSCLEROTIC ETIOLOGY

The second type of dissecting aneurysm has little in common with the classical type. It differs in etiology, location, age incidence, and mortality. Aneurysms of this type are the result of two manifestations of arteriosclerosis commonly seen in the aorta; *i.e.*, senile sclerosis of the media and atherosclerosis of the intima.

There has been considerable investigation into the nature of senile changes of the media. Wilens[46] observed that the ability of elastic fibers to recoil on relaxation is dependent upon their wavy or undulating structure, rather than on ability to stretch like a rubber band. His studies on the elasticity of the aortic wall indicate that with loss of elasticity there is a corresponding loss of the undulations in the elastic lamellas. This loss of elasticity was found to develop at a uniform rate for a given area of the aorta, and appears to affect all persons to approximately the same extent. With aging there is a constant, fairly uniform decline in elasticity. Further studies by Hass[19,20] demonstrate that with aging there is diffuse collagenous replacement of the interlamellar structures. This has a splinting effect on the elastic lamellas, so that in the dilated, sclerotic aorta the elastic network is always under tension.

Wilens found that the greatest loss of elasticity appeared at the bifurcation of the aorta and in areas most rigidly attached in situ, such as the portion of the descending aorta in contact with the spine. He noted that atheromatous degeneration occurred earlier in these areas than in the surrounding intima.

Blumenthal, Lansing and Wheeler[2] determined the amount of calcification of the media that occurs with senility by using microincineration techniques. They concluded that medial calcification increased with aging and hypertension, and found the greatest amounts near atheromas.

These diffuse changes in the media result in weakness and permanent dilatation of the aorta, and though due largely to aging, must be considered, along with the changes of atherosclerosis, as factors precipitating the dissection. Both types of changes are illustrated in Figure 4. The intimal lesions of atherosclerosis not infrequently ulcerate or calcify, and are usually surrounded by zones of extensive degeneration involving both elastic and smooth muscle components of the media. They are found in greater numbers in the abdominal aorta than in the thoracic portion and arch; when found in the arch, however, the lesions are most frequently in the region of the root. They are not confined to the intimal coat, but often involve a major portion of the media as well. Dissection and rupture may occur when the circulating blood gains access to the weakened media through these atheromatous ulcers and fissures.

Fig. 4. Arteriosclerotic aorta. There is straightening of the elastic lamellas, with collagenous replacement and hyalinization of interlamellar structures. The deep staining areas in the upper half represent collections of inflammatory cells, which are an incidental finding. Note the large atheroma below.

Mallory[30] has observed that when dissection does occur through such intimal lesions, the extent of the dissection is usually minimal; and if there is external rupture, it occurs near the primary intimal lesion. Twelve of the 15 cases of dissecting aneurysm autopsied in a ten year period at the State University of Iowa are believed to be arteriosclerotic in etiology, and all but 1 of these dissected through atheromatous ulcers or around calcified intimal plaques. This 1 case involved the distal 7 cm. of the abdominal aorta and the proximal right common iliac artery. It completely surrounded the aorta, and had ruptured neither internally nor externally.

A dissecting aneurysm of arteriosclerotic etiology is less likely to be fatal than one of the classical type. Eight of the 12 arteriosclerotic dissecting aneurysms studied here involved the lower abdominal aorta, and at autopsy were found to be incidental to the cause of death. The average age of these 12 patients was 69.6 years; the youngest, fifty-nine years of age, the oldest, eighty.

Only a small percentage of arteriosclerotic aneurysms occurs in the thorax; this is consistent with the distribution of atherosclerosis in the aorta. However, thoracic arteriosclerotic dissecting aneurysms may rupture externally into the same structures as classical dissecting aneurysms. In the abdomen, the dissection not uncommonly terminates with external rupture into the retroperitoneal tissues and the peritoneum. Extensive extravasation of blood into the mediastinum and pleurae has occurred when the rupture was high in the abdomen.

CLINICAL FEATURES OF DISSECTING ANEURYSMS

The onset of dissecting aneurysm is usually characterized by extremely severe, tearing, shocking pain which usually originates in the chest and may follow the course of the dissection into the abdomen. A direct relationship exists between the location of the pain and the site of dissection. The onset of dissection is often preceded by some activity which produces a sudden rise in blood pressure, such as a strain from lifting or at stool, or extreme emotional excitement.[39] Pallor, sweating and unconsciousness often follow in rapid order, and the patient may die immediately from rupture into the pericardial sac or from exsanguination into the chest or abdomen. Severe pain is not always present, however, and in some instances the initial symptoms occur while the patient is resting. If the dissection proceeds slowly, there is usually little if any accompanying pain. Occasionally the symptoms are effectively masked by those of another disease.

If the patient survives the initial attack, other symptoms will appear upon extension of the dissection. The blood pressure may rise again to higher than normal levels. Myocardial infarction, bundle branch block, and ventricular fibrillation have occurred when the dissection penetrated around one of the coronary arteries and diminished the size of its lumen.[45]

The pulses may be unequal in volume in opposite extremities, and if the dissection extends along a large vessel a double asynchronous pulse may be palpated.[33] In 1 case presented below, opposite carotid pulses were asynchronous. Elevated venous pressure may result from the pressure of hemorrhage into the mediastinum or pericardial sac. Visual disturbances and unconsciousness have resulted from extension of the dissection into the carotid arteries, with partial or complete occlusion of their lumens. Occlusion of the intercostal and lumbar arteries with resulting ischemia of the cord has been blamed for evanescent and bizarre neurological disturbances. Spontaneous periarterial sympathectomy produced by the dissection may cause sensations of heat although the body temperature remains normal. Occlusion of the subclavian or iliac arteries may be permanent and may result in gangrene of the extremities. When the renal and mesenteric arteries are involved, hematuria, melena, abdominal distention, and ileus may occur. Fever, leukocytosis and

anemia are common. Thrills and bruits have been frequently observed.[29]

The heart is usually enlarged, and signs of pericardial and pleural effusion may be found. Pleural effusion, when present, is usually on the left side.[39] The cause for this is not readily apparent, but may be due to the close anatomical relationship between the left pleura and the descending aorta. Blowing, rumbling murmurs are commonly heard over the supracardiac region. Electrocardiographic studies seem not to be diagnostic in dissecting aneurysm.[3] Compression and displacement of the esophagus and trachea may cause dysphagia, cough, cyanosis, and even suffocation.

When the dissection occurs in the abdominal aorta, the pain is usually localized to the abdomen, and is often accompanied by rigidity and tenderness. The aneurysm can often be palpated through the abdominal wall. Ecchymoses in the subcutaneous tissues of the flank, inguinal region, and scrotum have occurred following retroperitoneal hemorrhage.[32]

Dissecting aneurysms in the thorax may simulate myocardial infarction, cerebral embolism or hemorrhage, pulmonary embolism, peripheral vascular embolism or thrombosis, spontaneous pneumothorax, and mediastinal emphysema. In the abdomen they simulate nephrolithiasis or ureterolithiasis, ruptured hollow viscus, appendicitis, acute pancreatitis, and other abdominal inflammations, which may lead to surgical exploration.

ROENTGENOLOGIC FEATURES OF DISSECTING ANEURYSMS

Almost never is the diagnosis of dissecting aneurysm of the aorta made by the radiologist without recourse to the history and physical findings. Some roentgenograms are suggestive enough of this condition to warrant further investigation, but without a good history, the roentgen diagnosis of dissecting aneurysm is always difficult. There are findings, however, which justify the primary roentgen diagnosis of dissecting aneurysm. These will be discussed later.

A valuable aid in the diagnosis is a roentgenogram of the chest taken prior to the onset of the dissection. If such a film is available, comparative measurements of the supracardiac shadow and the diameter of the aortic knob may show an increase in their size which cannot be satisfactorily explained on any other basis than that of dissecting aneurysm. Of course, for such measurements to be valid, the roentgenograms must have been taken with comparable technique and with the patient in the same position. Often there is considerable change in the configuration and the width of the supracardiac shadow when the position is changed from erect to supine. Not as important, but still to be considered, is the phase of respiration. An attempt should therefore be made to have films taken in the same phase of respiration when these measurements are to be made.

Widening of the supracardiac shadow is most often seen in dissecting aneurysm of the classical type.[47] This widening may involve the entire thoracic aorta, or only a small segment of it. The widening may be accentuated by extravasation of blood into the mediastinum. Localized widening suggests not only the initial site of the dissection, but if it persists over a period of days or weeks, particularly if there is intimal calcification, it may also indicate that blood has entered a limited zone of dissection through an atheromatous ulcer or around the margin of an atheromatous plaque. With the more generalized idiopathic medial necrosis, the initial dissection tends to be fairly extensive.

If the patient survives the initial dissection, progress films of the chest may demonstrate extension of the process proximally or distally. Interest in obtaining a complete record of the course of the disease must be tempered by respect for the precarious physical condition of the patient. The shadow produced by an extension of the dissection along one or more of the large branches of the aortic arch is con-

sidered to be a pathognomonic sign.[47] As observed roentgenoscopically, the aneurysm may or may not pulsate. Evidence of left ventricular hypertrophy or generalized cardiac enlargement is usually present. Signs of pericardial effusion may be due to extravasation of blood into the pericardial sac.

As the essential feature of the disease is a dissecting hematoma which causes considerable thickening of the shadow of the aortic wall, conditions which enable one to measure the thickness of the wall contribute greatly to the accuracy of diagnosis. Such conditions occur occasionally in the progress of the disease. It has been observed that the dissected portion of the aneurysm is more radiolucent than the dense central core of the aorta,[25] particularly when the dissection involves only the lateral portion of the aorta with respect to the projection. The width of this radiolucent shadow corresponds closely to the thickness of the aortic wall (Fig. 10).

Calcification within the intima may delineate the inner margin of a dissecting aneurysm, as first demonstrated by Theodor Canigiani[5] in 1932. Atherosclerosis, the chief cause of intimal calcification, is often present in dissecting aneurysms, and syphilis, which causes intimal calcification in a younger group, is present in nearly 10 per cent of dissecting aneurysms.

Intimal calcification, if sufficiently dense, can be demonstrated roentgenographically throughout the entire circumference of the aorta, particularly if the background is of high radiolucency. More often, when the background is dense or the plaques are thin and lightly calcified, only calcification tangential to the projection is visualized. It is this tangential calcification that is of particular value in determining the thickness of the aortic wall. No problem is encountered in determining the outer margin of the aneurysm when its shadow lies in contrast to the radiolucent background of the pulmonary fields. It is apparent that in the chest optimal circumstances exist for the visualization of both intimal calcification and the lateral margin of the aneurysm. The normal aortic wall measures 2 to 3 mm. in thickness, and where thickening of the wall secondary to syphilitic aortitis occurs, or where there are superimposed shadows of pericardial or pleural reflections, measurements of 5 mm. are not uncommon. Measurements approaching 1 cm. suggest a dissecting hematoma between the arterial coats, and with 1 cm. or more, there can be little doubt of it.

When the background is of the same density as the aorta as in the abdomen, special procedures may be necessary to determine the outer limit of the vessel.

Retroperitoneal and intraperitoneal injections of air have been suggested by several investigators[8,9] to furnish a radiolucent background for the visualization of abdominal aneurysms of arteriosclerotic etiology, and this method should be equally useful in determining the presence of dissection within their walls. Barium within the esophagus, stomach, or bowel may also be used to furnish roentgenographic contrast. Displacement of abdominal structures, particularly the kidneys and ureters, is of considerable diagnostic importance.

In at least one instance, an old healed dissecting aneurysm has been demonstrated by the visualization of calcification within the new intimal lining of the dissection as well as in the intima lining the aortic lumen, thus producing an appearance of "double barrel" calcification throughout a major portion of the aorta.

Gillies[14] and Hampton[18] have independently observed that in many cases of dissecting aneurysm the notch below the aortic "knob" becomes obliterated. This change may also occur in syphilitic aneurysm, but the relative infrequency of this diagnosis today justifies stronger consideration of the dissecting type of aneurysm.

Recently, Paullin and James[35] have demonstrated dissecting aneurysm by the use of radiopaque contrast media within the aorta. It is probable that this technique will find extensive application in the future. Laminagraphic studies of the aorta have

also proved to be of assistance in determining the location and extent of aneurysmal dilatation.[42]

As has been emphasized above, the roentgen diagnosis of dissecting aneurysm has its limitations. If there has been extensive extravasation of blood into the mediastinum or pleurae, the aortic shadow may be entirely obliterated. Without films taken before the onset of the disease, it may be impossible to differentiate between dissecting aneurysm and extensive widening and tortuosity of the aorta due to arteriosclerosis or syphilis. When intimal calcification is present in either of the above instances, as is particularly likely in older age groups, the thickness of the wall is easily estimated and an intramural dissecting hematoma ruled out. Lateral and oblique films often give additional information, as well as demonstrate the nature of changes in the aortic configuration due to scoliosis and dorsum rotundum. Another difficulty may arise in the differentiation between the shadow of the superior vena cava and dissection along the innominate artery or the lateral margin of the ascending aorta.

Fig. 5. Case I. The right ureter and kidney have been displaced laterally by the dissecting abdominal aneurysm, the inner surface of which is indicated by the crescentic intimal calcification. The dissection lies between the intima and the tip of the right catheter. (The intimal calcification has been retouched for emphasis.)

REPORT OF CASES

Case I. *Entrance Complaint.* E. K., a woman, aged sixty-eight, was admitted because of severe right abdominal pain of three hours' duration.

Chronology of Symptoms. The onset of pain was sudden and knife-like, and became progressively worse. She experienced no nausea, hematemesis, hemoptysis or melena. She was known to have hypertension.

Physical Findings. Blood pressure, 110/90 mm. Hg. Forceful pulsations were palpated in the right mid-epigastrium; no bruit was heard in this region. Rigidity was noted in the right upper quadrant. The leukocyte count was 10,300. Kolmer and Kline tests were negative.

Roentgen Findings. Retrograde pyelography showed lateral displacement of the right kidney by a homogeneous mass containing crescentic calcification (Fig. 5).

Operative Procedure. A celiotomy was performed, and a large aneurysm of the abdominal aorta was found. The patient died twenty hours following admission.

Autopsy Findings. Examination through the operative incision revealed an aneurysm extending from just below the renal arteries to the bifurcation of the abdominal aorta. There was considerable retroperitoneal hemorrhage. There were many calcified plaques in the wall of the aorta. Dissection and external rupture had occurred on the right side. The autopsy diagnosis was arteriosclerosis and dissecting aneurysm of the abdominal aorta.

Comment. Although dissecting aneurysm was suspected clinically, the acute abdominal pain, rigidity, mild leukocytosis and occult hematuria suggested an inflammatory lesion, accounting for the exploratory procedure. This aneurysm is clearly of arteriosclerotic etiology.

Case II. *Entrance Complaint.* E. H., a man, aged seventy-four, entered the hospital in a state of shock.

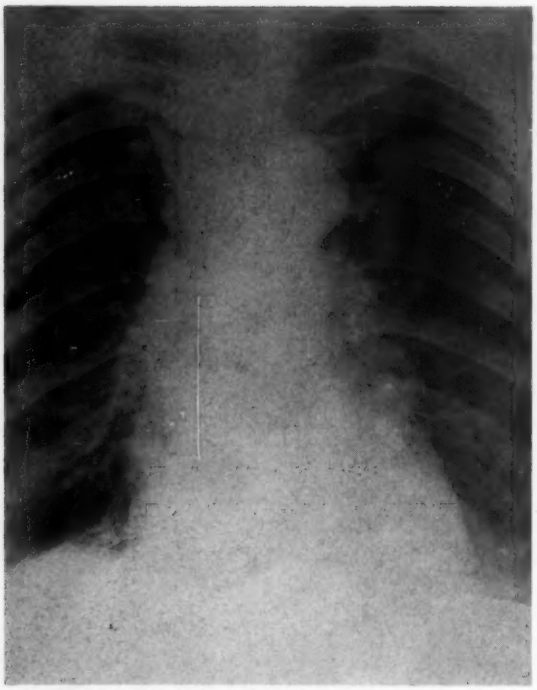

FIG. 6. Case II. The supracardiac shadow before the onset of dissection.

Chronology of Symptoms. While sitting in a railroad station, the patient had slumped over, "broken into a cold sweat," and vomited. On admission he was suspected of having a myocardial infarct. A film of the chest showed slight irregularity of the supracardiac aortic shadow, which was larger than that observed on a film taken two years before (Fig. 6 and 7). Kolmer and Kline tests were negative. The patient refused to remain in the hospital for further observation.

Ten weeks later he was brought in for examination after having been found lying in the street. After a febrile episode, he left the hospital a few days later against advice. He was admitted for the third time seventeen days later, again acutely ill with *Haemophilus influenzae* meningitis, and died on the twentieth hospital day.

Roentgen Findings. The history of acute chest pain and the increase in size of the supracardiac shadow led to a roentgenologic impression of dissecting aneurysm of the aorta. Subsequent films continued to show enlargement of the supracardiac shadow.

Autopsy Findings. There was a small dissecting aneurysm of the aortic arch, which had ruptured with extensive hemorrhage in the mediastinum. Dissection occurred around an atheromatous plaque in the intima (Fig. 8). Histopathologic examination showed extensive changes of arteriosclerosis.

Comment. This aneurysm is characteristic of the arteriosclerotic type, having originated around an atherosclerotic plaque, dissected a limited area, and ruptured externally near the original dissection.

CASE III. *Entrance Complaint.* J. N., a man, aged seventy, on admission complained of urinary retention.

Chronology of Symptoms. He had a two year history of urinary tract difficulties, and on admission had an indwelling catheter in his bladder. The initial hospital diagnosis was benign prostatic hyperplasia, cystitis and generalized arteriosclerosis.

Physical Findings. On examination, the heart was slightly enlarged. Blood pressure was 155/76 mm. Hg. Kolmer and Kline tests were negative. Ths hospital stay was marked by

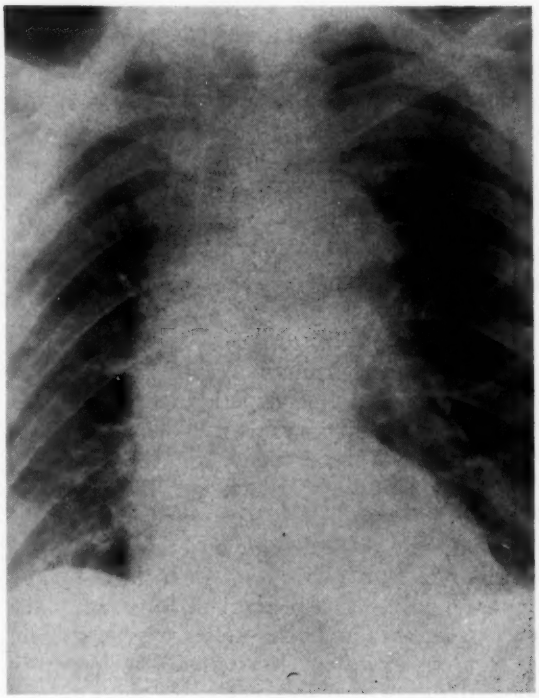

FIG. 7. Case II. The supracardiac shadow has become greatly enlarged following onset of dissection. There is no apparent change in cardiac size. The trachea is shifted to the right. The notch below the aortic "knob" is now absent.

febrile episodes, and hemolytic *Staphylococcus aureus* was recovered from the peripheral blood on several occasions. On the ninth hospital day the patient was found dead in bed, fifteen minutes after having returned from the bathroom.

Roentgen Findings. An admission film of the chest showed tortuosity of the thoracic aorta, calcification in the intima of the ascending aorta, and pulmonary emphysema (Fig. 9). A progress film of the chest demonstrated considerable cardiac enlargement and prominence of the root of the aorta. In comparison with the previous examination, there was increase in the thickness of the wall of the ascending portion of the arch, the measurement between the calcified intima and the outer surface of the aorta being 1.7 cm. (Fig. 10). The findings indicated a dissecting aneurysm of the aorta with rupture into the pericardial sac.

Autopsy Findings. Autopsy revealed a dissecting aneurysm of the ascending aorta which had ruptured into the pericardial sac. The sac was distended by 300 cc. of clotted blood. A thick layer of fibrinous exudate covered the pericardial and epicardial surfaces beneath the blood clot. Hemolytic *Staphylococcus aureus* was cultured from this exudate. The aortic

Fig. 8. Case II. A large irregular crater remains at the site of origin of the dissection. This was a partially calcified atheroma.

Fig. 9. Case III. Before the dissection. Note the prominence of the aortic root, with extensive calcification of the intima.

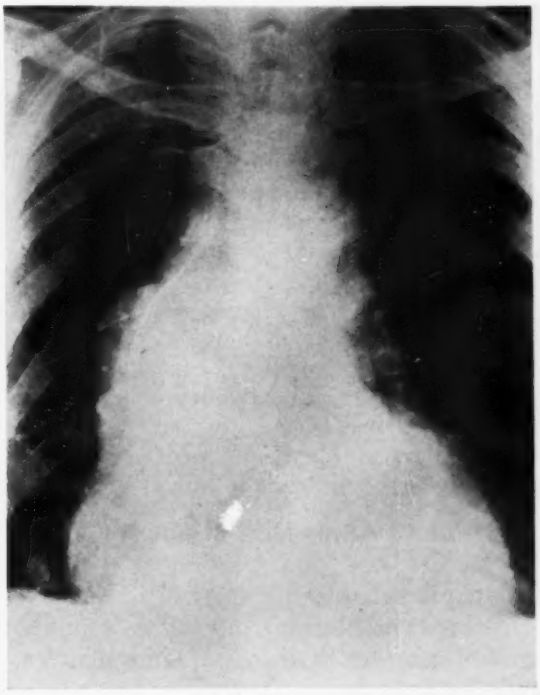

Fig. 10. Case III. Following dissection. The broad zone of relative radiolucency, lateral to the intimal calcification, represents the dissecting hematoma. Note also the enlargement of the cardiac shadow. (Calcification retouched for emphasis.)

FIG. 11. Case III. An irregular tear around a calcified atheroma on the posterior wall represents the site of origin of the dissection.

intima was thickened, and there were numerous large atheromatous plaques. A defect in the intima was found at the margin of such a plaque 6 cm. above the aortic valve leaflets (Fig. 11). There was acute cellulitis of the aortic wall at this point, and a thick layer of granulation tissue lined the aneurysm and blood-filled spaces outside the aorta. The communication between the dissecting aneurysm and the pericardium could not be definitely demonstrated. Histopathologic examination of the aorta showed collagenous replacement of the interlamellar structures with hyaline degeneration. The elastic lamellas had lost their characteristic undulations. There was extensive atheromatous degeneration of the intima (Fig. 4).

Comment. Although this aneurysm involved the ascending portion of the arch and ruptured into the pericardial sac, it originated in the margin of a calcified intimal plaque, dissected a limited area, and the underlying changes in the media were those of arteriosclerosis. It is interesting to note the extensive intimal calcification of the ascending aorta in the absence of history or serologic evidence of syphilis.

CASE IV. *Entrance Complaint.* M. F., a man, aged forty-six, entered the hospital complaining of pain in the chest.

Chronology of Symptoms. The patient was first seen in the out-patient clinic five months prior to this admission, with a complaint of dizziness and flatulence. A chest roentgenogram was reported as normal (Fig. 12). Kolmer and Kline tests were negative. His blood pressure was 210/140 mm. Hg.

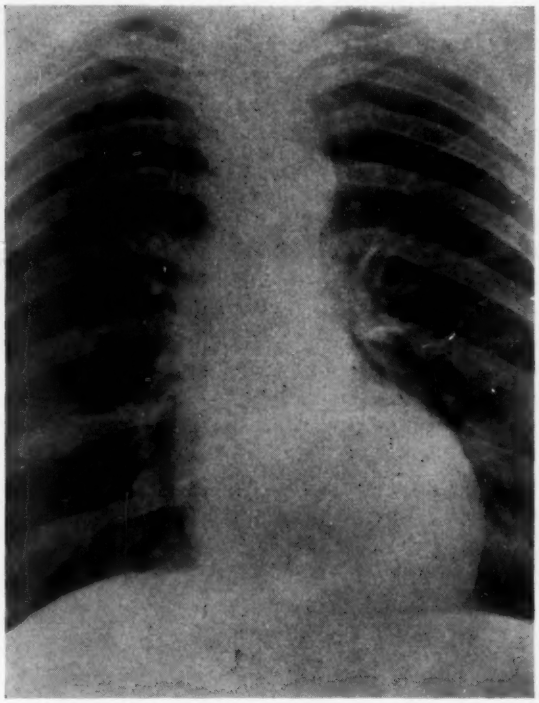

FIG. 12. Case IV. Posteroanterior roentgenogram taken at the time of the first examination. Note contour of the left ventricular hypertrophy.

Three days prior to this second admission, the patient, while dressing, had suddenly experienced excruciating pain in the back. Movement initiated a tearing pain in the chest, for which morphine was required. The patient's wife stated that she had noticed, prior to the onset of pain, that her husband's body became blue below the navel, and his genitalia turned black.

Dissecting aneurysm and an acute intra-abdominal episode were considered in the differential diagnosis.

Roentgen examination at this time showed widening of the supracardiac shadow and fullness of the descending aorta (Fig. 13 and 14).

The roentgenologic diagnosis was dissecting aneurysm of the aortic arch and descending portion.

After more than two months' stay in the hospital, he was discharged with the recommendation of bed rest. He was re-admitted eleven weeks later, following an attack of sharp pain in the left lumbar region. This was associated with hematuria and passage of fishworm clots. The blood pressure was still elevated, and early

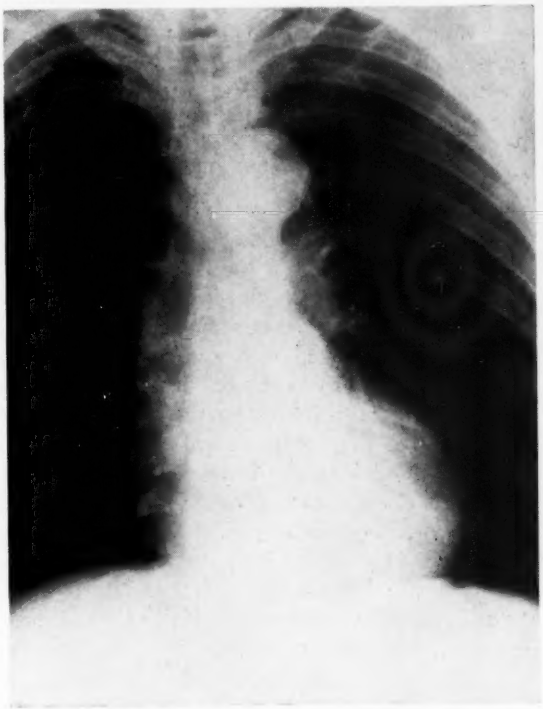

Fig. 13. Case iv. Roentgenogram taken soon after the initial dissection. Note the widening of the supracardiac shadow, and increase in the thickness of the arch of the aorta.

papilledema and hypertensive retinopathy were noted. He became comatose and died 162 days following the initial attack.

Roentgen Findings. The first chest roentgenogram demonstrated only slight prominence of the left ventricular contour. It did furnish, however, a basis for comparison with the second examination. The findings immediately after the dissection were not in themselves sufficiently striking to warrant a diagnosis of dissecting aneurysm, but in comparison with the first film the change was obvious. Later in the course of the disease, the aneurysmal dilation became pronounced (Fig. 15).

Autopsy Findings. Autopsy disclosed severe

Fig. 14. Case iv. Left anterior oblique barium esophagram, showing widening of the aorta throughout its intrathoracic course.

intimal atherosclerosis and three separate aneurysms of the aorta. The first was saccular, approximately 3 cm. in diameter, and was just distal to the innominate artery. The opening into the aneurysm was slit-like, and measured less than 2 cm. in length (Fig. 16). The second was 4 cm. long, and was located at the junction of the arch and descending aorta. It consistuted a definite splitting of the media with a re-entry into the aorta, so that a bridge of aortic tissue was left across the site of the aneurysm. A similar aneurysm was present above the renal arteries.

Histopathologic examination of the aorta showed evidence of medial necrosis of moderate degree, corresponding to the changes seen in Type 1 as described by Sailer. In some areas there was evidence of considerable attempt at repair.

Comment. This is a typical case of classical dissecting aneurysm, in which re-entry occurred and was followed by partial healing. Although it was suspected clinically, the roentgen findings made the diagnosis of dissecting aneurysm conclusive.

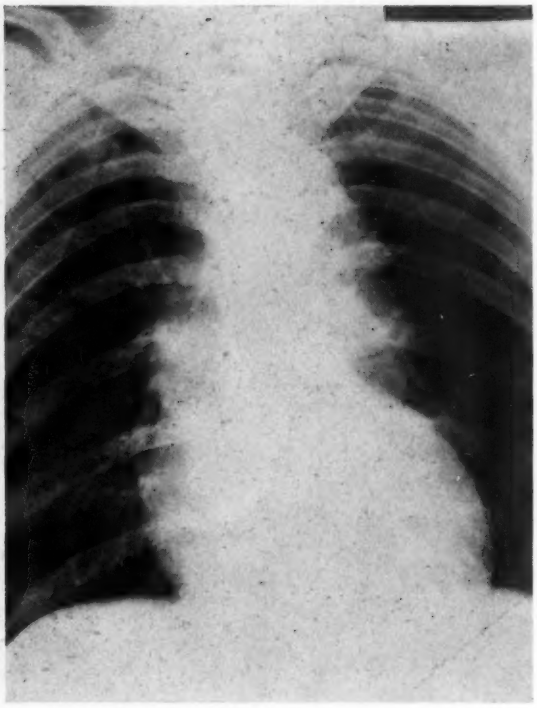

FIG. 15. Case IV. Roentgenogram taken shortly before death. The aortic shadow is now conspicuously widened.

CASE V. *Entrance Complaint.* J. J. S., a male, aged twenty-seven, entered the hospital complaining of pain in the right anterior portion of the neck, difficulty in swallowing, weakness, and fever of four days' duration.

Chronology of Symptoms. The patient had spent three years of unlimited active duty in the Army. Four days before admission he had, while walking, experienced a sudden sensation of tightening and sharp, severe pain in the front of his neck, followed in the next half hour by similar attacks. The last was most severe.

The next four days were spent in and out of bed at home, with mild fever and dysphagia.

Physical Findings. A murmur was heard over the aortic valve region, and sharp tenderness was present over the right thyroid area. The patient's temperature was 101.4° F. The blood pressure in the right arm was 112/68 mm. Hg., and in the left was 150/72. Pulsations were visible in the neck, and the carotid pulses were asynchronous, the left appearing before the right. The left radial pulse was strong and full; the right, feeble. A to-and-fro murmur was heard over the base of the heart, and was transmitted into the neck, but the heart was not clinically enlarged. Examination of the abdomen was negative. The leukocyte count ranged from 11,000 to 13,000 per cu. mm. Kolmer and Kline tests were negative. The clinical diagnosis was dissecting aneurysm of the arota.

Roentgen Findings. In a chest roentgenogram, the lung fields were clear and the heart and great vessels were within normal limits. There was no significant enlargement of the supracardiac shadow (Fig. 17). However, roentgenoscopy of the chest with barium esophagram showed enlargement of the aorta in the ascending portion and the arch and left ventricular hypertrophy (Fig. 18).

Hospital Course. The first four days were uneventful, except for persistent fever of from 99.6° to 102.6° F. During the early morning of the fifth hospital day he quietly died.

Autopsy Findings. Autopsy revealed an extensive dissecting aneurysm of the aorta, which had dissected into the pericardial sac, causing death by cardiac tamponade. The initial rupture was a transverse tear about 1 cm. above the aortic valve, and the dissection extended into the innominate, right subclavian and right carotid arteries, with a small exit rupture in the carotid. The dissection extended into the abdominal aorta, stopping just short of the bifurcation. The media was extremely friable, separating with the slightest trauma. The site of rupture into the pericardial sac could not be definitely demonstrated.

Histopathologic examination of the aorta showed evidence of medial necrosis of moderate

FIG. 16. Case IV. The slit-like opening on the left represents the mouth of the saccular aneurysm. Note the extensive dissection to the right in the illustration.

degree, corresponding to the changes seen in Type 4 as described by Sailer.

Comment. The clinical findings in this case were so conclusive that the chest roentgenograms gave mere confirmatory evidence. The asynchronous pulses in the carotid arteries are unusual. This is a typical case of the classical type.

Case vi. *Entrance Complaint.* G. W. W., a man, aged twenty-four, complained of palpitation of the heart and weakness of six weeks' duration.

Chronology of Symptoms. Since the age of twelve years he had been more easily fatigued than his companions. There was no history of rheumatic fever or congenital cardiac disease.

Six weeks before admission, he developed severe substernal pain, which lasted for twenty-four hours. He was confined to bed for more than a week.

Physical Findings. The patient did not appear to be acutely ill. The left pupil was larger than the right. Bilateral carotid pulsations were present, but the right was weak to palpation. A harsh systolic blow accompanied by a thrill was heard over the left carotid. The trachea was deviated 2 cm. to the right. There was pronounced dullness beneath the manubrium sterni, and this region heaved with the hearbeat. A to-and-fro murmur was heard in the aortic area. The heart was diffusely enlarged, and the pulse was 100 per minute. The radial pulse could not be palpated in the right arm, but was full and of celer contour in the left. The blood pressure in the right arm was 54/40 mm. Hg.; in the left, 115/30. The systolic pressure in the left leg was 95. Durozier's sign was present. There was a fever of less than one degree. The leukocyte count varied from 14,000 to 15,000 per cu. mm. Repeated blood cultures showed no growth, and electrocardiograms were reported normal. Kolmer and Kline tests were negative.

Fig. 18. Case v. Left anterior oblique barium esophagram. Note the dilatation of the aortic arch.

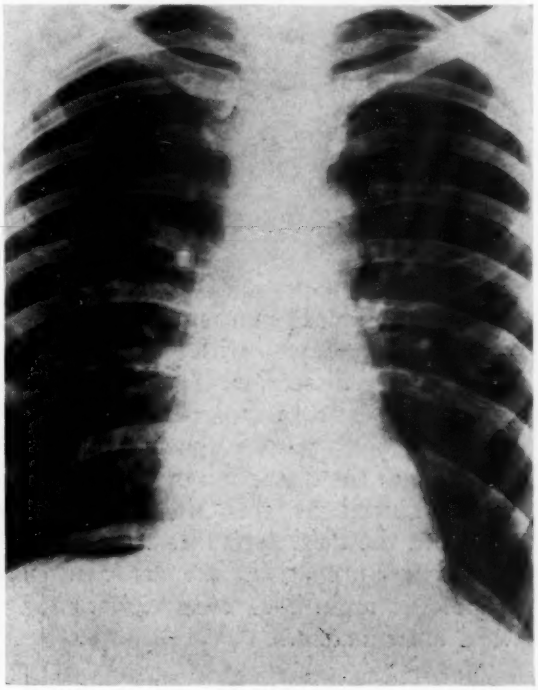

Fig. 17. Case v. Posteroanterior roentgenogram following initial dissection, with only minimal widening of the arch. The notch is absent below aortic "knob."

Hospital Course. On the evening of the fourth hospital day, the patient experienced an attack of sharp, nonradiating, continuous precordial pain. He developed a persistent cough and expectorated frothy, blood-tinged sputum. Although the pulse rate was elevated, and a chest film showed an infiltration in the left lower lung field, the temperature remained

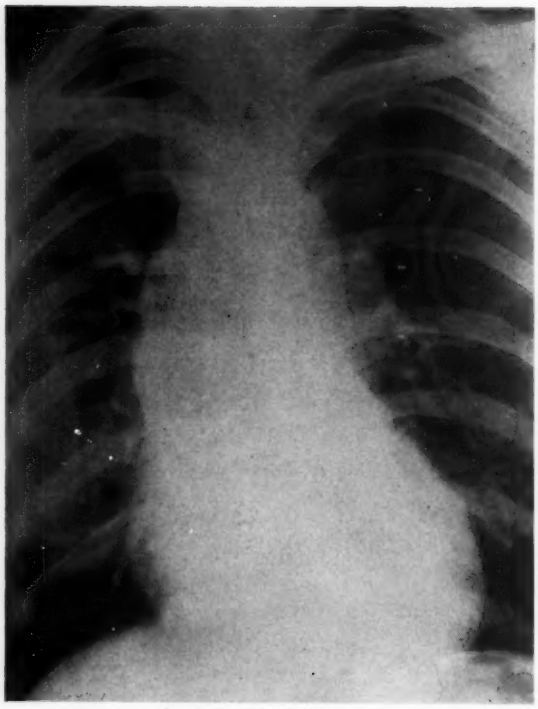

FIG. 19. Case VI. Posteroanterior admission roentgenogram, demonstrating moderate widening of the supracardiac shadow, and cardiac enlargement. Note absence of notch below aortic "knob."

within one degree of normal. A similar attack occurred on the twenty-fourth hospital day. Following this the patient slipped slowly into cardiac failure, and died suddenly on the thirty-ninth hospital day.

Roentgen Findings. An admission film of the chest showed slight but definite enlargement of the heart. The aortic arch was widened (Fig. 19). Cardiac roentgenoscopy showed hypertrophy of both ventricles. There was dilatation of the ascending and transverse portions of the aortic arch with expansile pulsation. The barium-filled esophagus was displaced posteriorly by the aorta and the posterior border of the heart. A film of the chest taken following the first attack of severe precordial pain showed a smaller supracardiac shadow than had previously been observed.

Autopsy Findings. At autopsy there was a large dissecting aneurysm of the aorta which extended from the aortic ring to the bifurcation. The dissection also extended along the innominate artery, producing severe narrowing of its lumen. There was one 5 mm. tear through the intima 6 cm. above the aortic ring, and numerous other small communications were present between the aorta and the aneurysm along the course of the aorta. There was no evidence of rheumatic or congenital cardiac disease. Histopathologic examination of the aorta showed evidence of medial necrosis of severe degree, corresponding to the changes seen in Type 4 as described by Sailer. There was extensive accumulation of mucoid material.

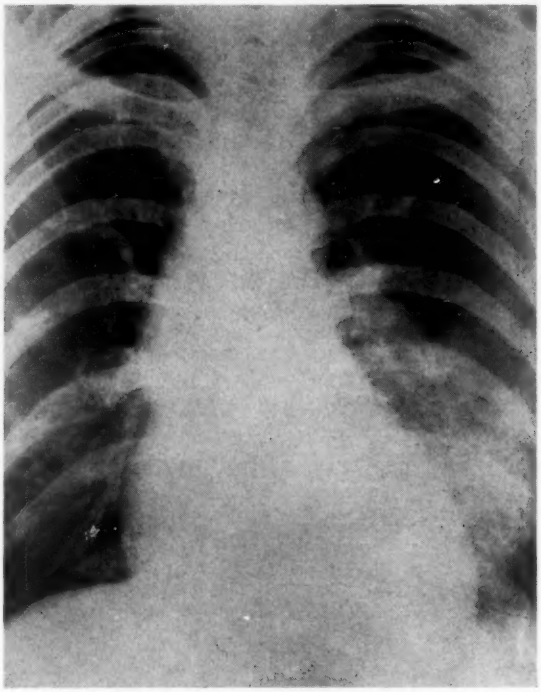

FIG. 20. Case VI. Progress roentgenogram. The supracardiac shadow is greatly reduced in width following extension of the dissection.

Comment. This is another case of classical dissecting aneursym, with partial healing following the dissection. The reduction in the size of the supracardiac shadow following the second attack is most unusual. An extension of the dissection or re-entry may have occurred at this time, causing a change in the fluid dynamics with diminution of pressure within the dissected portion of the arch. This case was not diagnosed antemortem.

DISCUSSION OF CASES

Of the 6 cases of dissecting aneurysm presented above, 3 were of the classical type, and originated in the ascending por-

tion of the aortic arch. Idiopathic medial necrosis was the underlying lesion. The average age of these 3 patients was 32.6 years. The cases are typical in that the intimal ruptures were in the ascending aorta, occurred transversely, and did not originate in atheromatous plaques. These aneurysms dissected extensively and involved the abdominal as well as the thoracic aorta. Re-entry occurred in all 3 cases. This may account for the extended periods of survival of 2 of the patients, though the third lived for only eight days.

The dissection extended along the innominate artery in 2 of the patients, producing reduction of blood pressure in the right arms of both, and asymmetry of the carotid pulses in one. Two of the 3 had questionable hypertension after the dissection, and so far as is known, had none before dissection occurred. The third had a long history of severe hypertension, which persisted through repeated dissections.

The observation of cyanosis of the lower body and extremities in 1 case is most unusual, and furnishes additional insight into the mechanism of dissection. One can visualize the forceful stream from the left ventricle, directed by the projecting margin of the torn intima into the medial split, dissecting and tearing through the weakened coat and eventually closing the aortic lumen by compressing the stripped intima against the opposite wall. Such occlusion would not be relieved until the dissection had reached some firm barrier and re-entry had occurred.

Death in 1 case was due to rupture into the pericardial sac, with cardiac tamponade. The other 2 cases died of cardiac failure.

Of the 3 aneurysms of arteriosclerotic etiology, 2 occurred in the thorax and 1 in the abdomen. Both aneurysms of the arch originated in calcified intimal atheromas. Although it is not known for certain, the abdominal one probably did so as well. None of the 3 dissected extensively, and none extended along the branches of the aortic arch.

The average age of the 3 arteriosclerotic cases was over seventy years. Two died of external rupture and exsanguination; the third of rupture into the pericardial sac with cardiac tamponade, a mechanism most often seen in the classical type.

Following Ponseti's experimental productions of scoliosis and dissecting aneurysm in rats, these cases were checked for scoliosis. In 2 of the 3 patients dying of classical dissecting aneurysm, mild dorsal scoliosis was found. Severe dorsal scoliosis was recorded in the autopsy or roentgen examination of other cases not reported here in detail.

SUMMARY

1. A review of the literature and study of fifteen autopsied cases of dissecting aneurysm have led to the conclusion that there are two different types of dissecting aneurysm: one due to extensive medial necrosis, and the other secondary to arteriosclerosis and senile changes in the aorta. These two types differ in location and extent of dissection, age incidence, and mortality.

2. The diagnosis of dissecting aneurysm can rarely be accomplished by roentgen findings alone, but requires close correlation of history, physical findings, and roentgen examination.

3. Sudden increase in the size of the supracardiac aortic shadow or a demonstrable increase in the thickness of the aortic wall are reliable signs of dissecting aneurysm. Six cases are presented to demonstrate these and other signs.

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THE ROENTGEN DIAGNOSIS OF BLEEDING LESIONS OF THE SMALL INTESTINE*

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BLEEDING from the gastrointestinal tract, with or without pain or other symptoms, is a common manifestation of organic disease. The search for the cause of bleeding usually requires roentgen examination of one or more portions of the digestive tract. Although the cause may be readily located, it is sometimes very difficult to disclose.

Bleeding may be so slight as to be detected only by chemical tests of the feces, or it may be massive, resulting in a sharp reduction in the hemoglobin, requiring transfusion. Single massive hemorrhages occasionally occur for which a satisfactory explanation cannot be found. Inasmuch as they are not repeated, they remain a medical mystery. Repeatedly positive tests for occult blood in the stool, or recurrent gross hemorrhage, on the other hand, require explanation and demand repeated searches until the cause is found.

The great majority of bleeding lesions of the gastrointestinal tract will be found in esophagus, stomach, duodenum, or colon. Smith, Good and Gray[14] reported that 95 per cent of cases with melena or hematemesis of noncolonic origin would be found in the upper gastrointestinal tract (duodenum or above). This places the incidence of small intestinal sources of noncolonic bleeding at about 5 per cent.

Partly because of this low incidence of lesions, and partly because of the difficulty in studying it, the small intestine is often neglected or inadequately examined. In the literature are numerous comments upon the low percentage of small intestinal neoplasms detected roentgenologically. Boman,[1] for instance, reported that only 25 per cent of jejunal carcinomas can be diagnosed by the roentgen ray. We feel that systematic handling of patients with unexplained gastrointestinal bleeding will result in the demonstration of small intestinal lesions in a higher percentage of cases. Such demonstration may require repeated examinations, frequently with intestinal intubation. Weber and Kirklin[16] reported that the small intestinal pathology was detected in 89 per cent of patients examined roentgenologically. Their material included duodenal neoplasms. They felt that the high percentage of diagnostic examinations was due to the improved methods of studying the small intestine in recent years. They did not use intestinal tubes in their studies.

The purpose of this paper is to present a study of bleeding lesions of the small intestine which were seen at the Presbyterian Hospital in New York between 1930 and 1950. The medical records of 72 patients with verified lesions of the small intestine were reviewed.

MATERIAL

Only lesions of jejunum and ileum were included in this study. Lesions of the duodenum were omitted because the examination of this region should be routinely part of the study of the upper gastrointestinal tract. Lesions of the ileocecal valve were excluded because they are usually demonstrable on examination of the colon. Cases in which there were other lesions to which bleeding might be ascribed, and metastatic neoplasms, were also excluded.

Of the 72 patients with lesions of the jejunum and ileum, 53 proved to have neoplasms and 19 inflammatory lesions. The type of diseases encountered in these cases

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Table I

SEVENTY-TWO VERIFIED LESIONS OF JEJUNUM AND ILEUM

Neoplasms			
Malignant		Benign	
Adenocarcinoma	18	Leiomyoma	4
Carcinoid	9	Lipoma	5
Lymphosarcoma	6	Fibroma	3
Leiomyosarcoma	3	Adenoma	1
Plasmocytoma	1	Hemangioma	1
Unclassified	1	Lymphangioma	1
Non-neoplastic Lesions			
Tuberculous enteritis			3
Regional enteritis			11
Miscellaneous			4
Meckel's diverticulum		1	
Heterotopic gastric mucosa		1	
Endometriosis		1	
Acute jejunal ulcers		1	

is shown in Table I.

None of the four lesions listed under "miscellaneous" had preoperative roentgen studies, although all had intestinal bleeding. They will not be included in subsequent statistics. Additional causes of intestinal bleeding, not usually treated by resection, but which should be kept in mind, are purpura, radiation enteritis, diverticulosis and diverticulitis, leukemia, sarcoidosis, and abdominal aneurysm perforating into the intestine.

Not all of the 72 patients had been studied adequately either clinically or roentgenographically prior to surgery. Only 51 cases had adequate preoperative roentgen studies and stool examinations for blood. Of these 51 cases, 37 were neoplasms and 14 inflammatory lesions. In this group 37 manifested intestinal bleeding and 4 had anemia without melena.

The incidence of the chief symptoms of organic disease of the small intestine, namely, bleeding, diarrhea, and pain, is shown in Table II.

The 3 cases of tuberculous enteritis all had bleeding, but only 1 had diarrhea. This is a very small series because it is limited to lesions which involved only the small intestine. Because this seemed to give a misleading incidence of symptoms, we reviewed the cases of tuberculous enteritis involving both small and large intestine and found that, of 19 cases, there had been bleeding in 14, pain in 14, and diarrhea in 13. The bleeding had been gross in 5 instances and occult in 9 instances. In this group there was no significant difference in the incidence of diarrhea, pain and bleeding.

In the neoplastic group bleeding was the most common symptom. Pain was frequent but diarrhea was relatively rare. In regional enteritis the reverse was true, pain and diarrhea being the common symptoms and bleeding relatively rare.

The character of the bleeding in the benign neoplastic lesions was gross in every instance, but in the malignant neoplasms there were 6 patients who had occult bleeding. The number of transfusions given some of these patients before the diagnosis was established is surprisingly high. One woman received thirty transfusions, another sixteen, and several patients had had as many as ten.

All the 51 patients had roentgen examination of either the upper gastrointestinal tract or of the colon, and usually both, prior to the small intestinal studies. The small intestinal studies were conducted by several methods:

1. The routine small intestinal study after oral administration of 4 oz. of barium sul-

Table II

	Total	Bleeding	Pain	Diarrhea	Anemia without Bleeding
Malignant neoplasms	27	22	12	4	0
Benign neoplasms	10	6	7	1	2
Regional enteritis	11	3	9	6	2
Tuberculous enteritis	3	3	2	1	0

fate suspended in 5 oz. of normal saline. Films were taken at half hour intervals and roentgenoscopy done as indicated by wet film inspection of these half-hour films.

2. Barium injected through an intestinal tube. These intubation studies were done in three different ways: (a) The small intestinal enema as described by Schatzki. (b) In the presence of mechanical ileus, a Miller-Abbott tube was passed to the obstruction, the intestine deflated, and barium injected through the tube and studied roentgenoscopically. (c) In the presence of continued bleeding unexplained by barium studies, a tube was passed into the intestine and specimens of intestinal content aspirated as the tube advanced each foot. At the level at which a guaiac positive specimen was obtained, barium was injected through the tube and studied roentgenoscopically.

Demonstration of the lesion usually, but not always, makes it possible to determine whether it is neoplastic or inflammatory. The neoplastic lesions are either intraluminal filling defects or relatively short, rigid constrictions of the lumen with abrupt margins and destruction of mucosa. The inflammatory lesions are likely to be longer, often multiple, and the margins are more tapered in appearance than are neoplasms. However, granulomatous masses may simulate exactly a neoplasm and, conversely, neoplasms are sometimes long and multiple and may simulate inflammatory disease.

The demonstration of the site of bleeding is of prime importance. The differentiation between neoplasms and inflammations is desirable, but of secondary importance. If it appears that the disease is or could be neoplastic, operation is mandatory.

TABLE III

	Total	Detected by Roentgen Ray
Malignant neoplasms	27	25
Benign neoplasms	10	7
Tuberculous enteritis	3	2
Regional enteritis	11	9
	51	43

In this series of 51 patients the lesion was demonstrated in 43 instances (Table III).

The type of examination by which the lesion was demonstrated is shown in Table IV.

TABLE IV

	Detected by		
	Routine Small Intestine	Small Intestine Enema	Localized Tube Study
27 malignant neoplasm	17	1	7
10 benign neoplasm	6	0	1
3 tuberculous enteritis	2	0	0
11 regional enteritis	8	0	1

These figures give an indication of the frequency with which intubation studies were used in demonstration of these lesions. It should be noted, however, that in several instances in which the presence of the lesion was confirmed by an intubation study, and is therefore credited to the tube study, the abnormality had first been noted on routine small intestinal study, and could probably have been confirmed by re-examination with ingested barium.

The two principal causes of failure to demonstrate lesions by routine small intestinal study are:

(1) Disregard of slight alterations in pattern of small intestinal loops.
(2) Uneven or irregular distribution of the barium due to flocculation or to abnormal motor function of the small intestine.

Obviously only meticulous attention to detail will eliminate the first error. The second difficulty can be partly avoided by use of colloidal preparations of barium which may give a more uniform distribution of the opaque medium in the small intestine (Fig. 1). All of the cases in this study were examined with barium sulfate suspended in normal saline. We have not

Fig. 1. Hodgkin's Disease of Ileum. (*A*) Small intestinal study using barium sulfate in saline shows irregular distribution of the barium and does not demonstrate with certainty the segment of ileum involved with Hodgkin's disease. (*B*) Small intestinal study one month later using a colloidal barium preparation shows more uniform distribution of barium, and demonstrates a segment of ileum in the left lower quadrant which shows irregularity in contour and destruction of mucosal folds. This case was not included in the series because the disease was not primary in the small intestine.

as yet had sufficient experience with colloidal barium to warrant a statement concerning its merit, but it seems probable that it may be a more satisfactory medium to use.[17] Regardless of the medium used, it is extremely important to evaluate the small intestinal study on the basis of distribution of barium when searching for organic disease. If the distribution is not uniform, study should be made by an intubation method (Fig. 2).

REPORT OF CASES

The following cases are illustrative of the group:

Case 1. A fifteen year old white man was admitted in May, 1946, because of severe anemia, the hemoglobin being 5 gm. per 100 cc. Three years previously he had had two episodes of abdominal pain and passed large quantities of bright red blood per rectum. He had been admitted to a hospital at this time, and his gastrointestinal tract was studied by roentgen ray, but no diagnosis was made. He was given several transfusions and discharged. A third hemorrhage occurred seven months prior to admission to Presbyterian Hospital, and he had continued to bleed intermittently in the interval.

Small intestinal study revealed an oval translucent shadow 2 by 3 cm. in the wall of the lower ileum (Fig. 3, *B* and *C*). Transient intussusceptions of the mass were noted. The softness and uniform translucency suggested a lipoma.

At operation a pedunculated mass 2.5 cm. in diameter was found in the lower ileum. An associated intussusception was reduced. A small ulcer at the tip of the tumor accounted for bleeding. The histopathologic diagnosis was lipoma of the ileum.

It is noteworthy that the first two hemorrhages in this case were bright red blood, not tarry stools. The color of the blood is more dependent upon the rate of bleeding than upon the level of bleeding. Large amounts of blood, even high in the intestinal tract, have a rapid transit time due to bulk and thus the color is not changed.[7]

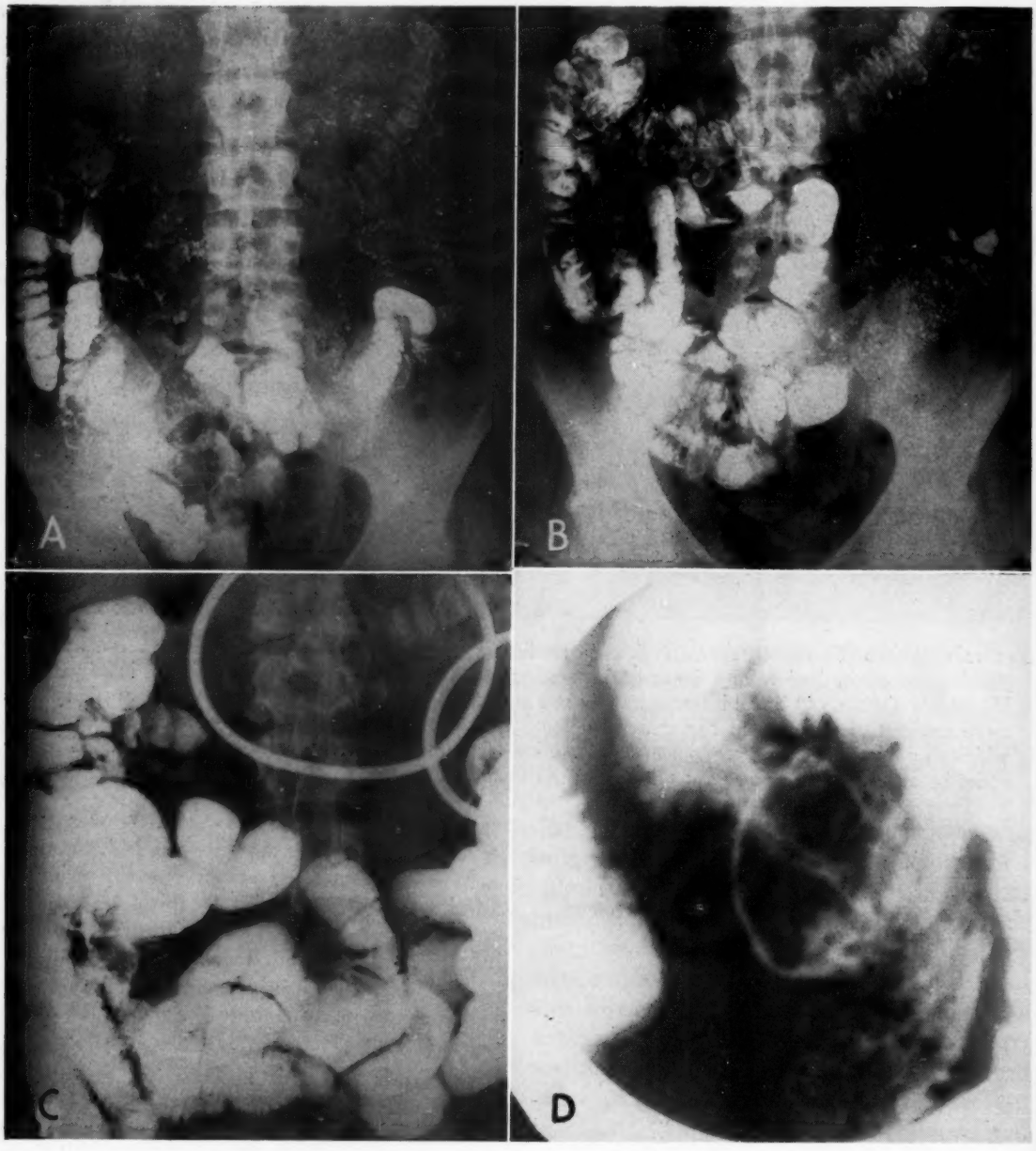

Fig. 2. Carcinoid of Ileum. (*A*) and (*B*) Films of a small intestinal study show no evidence of tumor, but there is irregularity in the distribution of the barium. This study is therefore not entirely satisfactory for exclusion of organic disease. (*C*) Filling of jejunum and ileum by a small intestinal enema revealed an abnormality in contour of a loop of the lower ileum in the lower right abdomen. (*D*) Pressure film shows a ring-shaped barium shadow suggesting barium surrounding an intraluminal mass.

Case II. A forty-six year old white woman was admitted because of four weeks of epigastric pain, nausea, vomiting, and fatigue.

Physical examination and laboratory studies revealed no positive findings except guaiac positive stools. She had no anemia. At this first admission she had roentgen examinations of stomach, duodenum, and colon, pyelograms and sigmoidoscopy, but no small intestinal study.

The complaints were attributed to a functional disorder and she was discharged.

One month later she was readmitted for "tiredness." This time, in addition to guaiac positive stools, she had an anemia (red blood

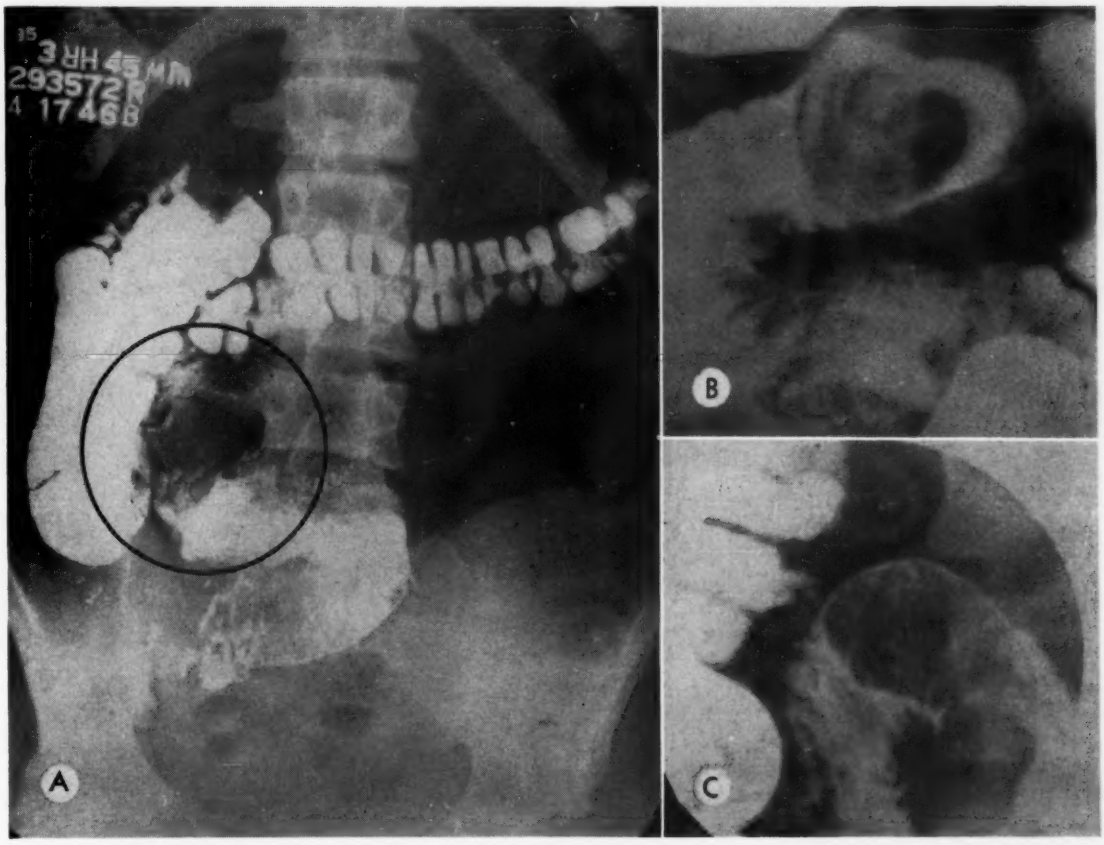

Fig. 3. Case I. Lipoma of Ileum. (*A*) A film from the small intestinal study shows the area in which pressure films were taken. (*B*) and (*C*) Pressure films of lower ileum show an ovoid radiolucency in the lower ileum.

cell count 3,320,000). A small intestinal study showed a localized irregularity in a loop of intestine in the left lower quadrant, visible on only one film of the series (Fig. 4, *A*). A second small intestinal study was done two days later, and roentgenoscopic examination was carried out when the loops of intestine in the left lower quadrant were filled. A short, rigid narrowing of the lumen with destruction of mucosa was demonstrated roentgenoscopically and on pressure films (Fig. 4, *B* and *C*).

This was a moderately well differentiated colloid type carcinoma of the jejunum with beginning extension into the mesentery. It was excised and the patient is well two and one-half years later.

Case III. A thirty-eight year old woman was admitted to a hospital in March, 1950, because of weakness, palpitation, dyspnea and nausea. During the previous twenty-four hours she had passed five or six loose, tarry stools. Hemoglobin on admission was 25 per cent, and she was given three transfusions.

Subsequent studies included roentgen examination of the upper gastrointestinal tract and colon, and proctoscopic examination, all of which were normal. She was discharged without a diagnosis.

One month later she was readmitted for weakness and anemia, and given another transfusion. Because she developed a transfusion reaction she was transferred to Presbyterian Hospital. At this time the stool specimens were strongly guaiac positive. An upper gastrointestinal examination, barium enema, double contrast enema, and routine small intestinal study were done, and were found normal. Since the stools were guaiac positive at this time, a tube was passed until a positive specimen was obtained, and barium was then injected through the tube. Since the tube was assumed to have passed the site of bleeding by

FIG. 4. CASE II. Adenocarcinoma of Jejunum. (*A*) The two hour and fifteen minute film of a routine small intestinal study shows a change in the pattern of a short segment of intestine in the left lower quadrant. (*B*) and (*C*) Pressure films taken during a repeat small intestinal study two days later show a localized irregular narrowing of the lumen, and mucosal destruction.

the time a guaiac positive specimen was obtained, the balloon was inflated to block the lumen so that the injected barium would fill the lumen proximal to the balloon. A circumscribed circular defect was found in the lower ileum (Fig. 5, *B*).

This proved to be a carcinoid. Two smaller tumors were found in the adjacent ileum, and there were metastases to the mesenteric nodes. Following resection, she was symptom free for a year and a half, but in the past month she has had symptoms suggesting recurrence.

In retrospect the first small intestinal study showed a circular radiolucency in the lower ileum which was undoubtedly due to the tumor (Fig. 5, *A*).

CASE IV. A fifty-seven year old white woman was admitted because of abdominal pain of five years' duration, and loss of 10 pounds in the preceding six months. She had had one episode of red blood in the stools. Physical examination revealed a tender mass in the left abdomen, below the umbilicus. A small intestinal study which had been done elsewhere was presented, and showed a short segment of narrowed jejunum, in which the mucosa had been destroyed. This was confirmed by a repeat small intestinal study (Fig. 6).

At operation an adenocarcinoma of the jejunum was resected. She is well six years later.

SUMMARY

In the presence of gastrointestinal bleeding, the upper gastrointestinal tract and the colon should be investigated first, but when these examinations do not reveal the cause of bleeding, the small intestine must be examined. The paucity of physical

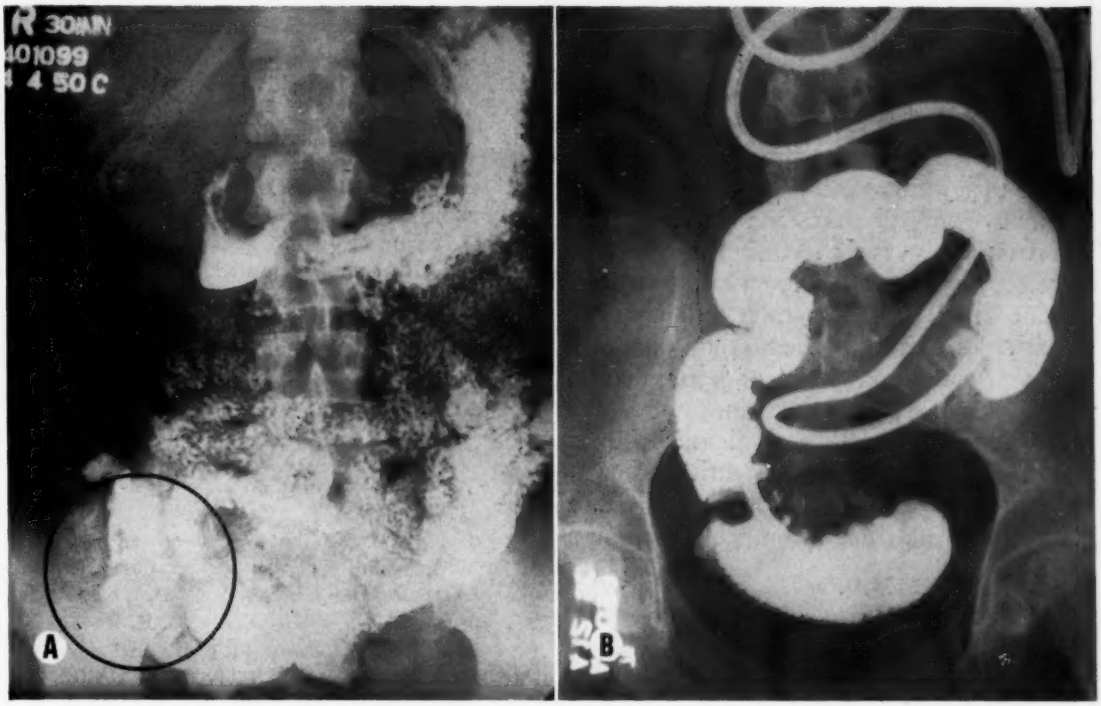

FIG. 5. Case III. Carcinoid of Ileum. (*A*) The thirty minute film of a routine small intestinal study shows a rounded radiolucency in a loop of lower ileum. This was demonstrated on only one film and not noted roentgenoscopically the time of this examination. (*B*) Eight days later a Miller-Abbott tube was passed to a point at which a guaiac positive specimen was aspirated. Barium injected through the tube demonstrated a spherical mass in the ileum.

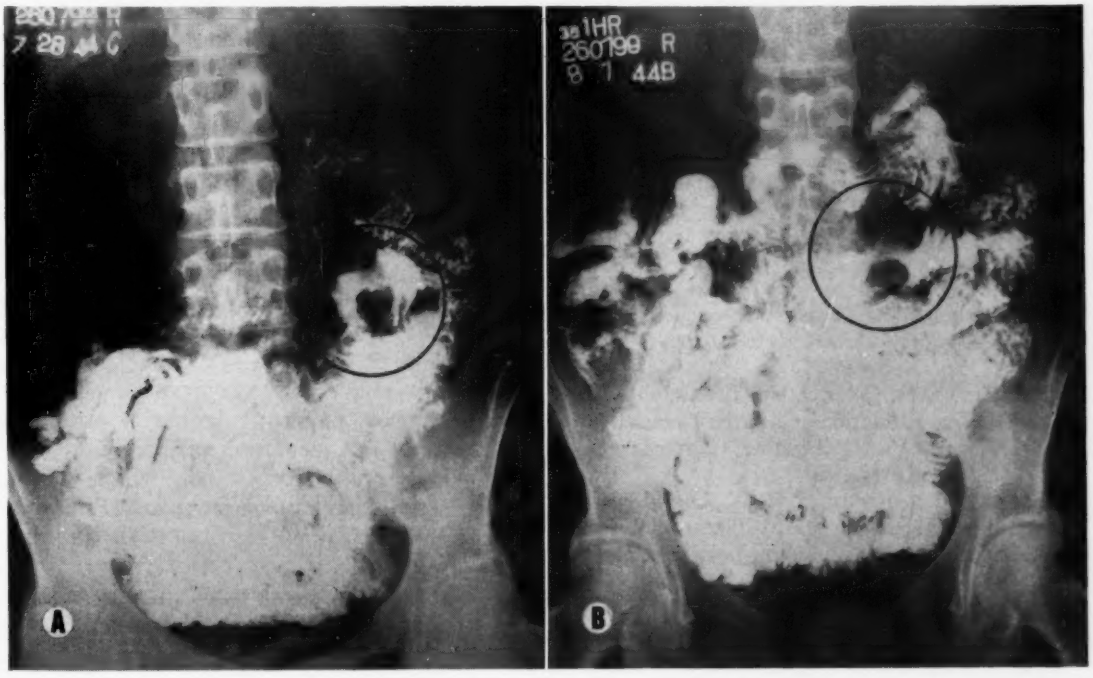

FIG. 6. Case IV. Adenocarcinoma of Jejunum. (*A*) A film from the small intestinal study of July 28, 1944, showed an irregularly narrowed segment of jejunum, devoid of mucosal markings, in the left mid-abdomen. (*B*) A repeat small intestinal study on August 7, 1944 reproduced the abnormal segment of jejunum previously demonstrated.

findings should not prevent examination of the small intestine in patients who have either bleeding or anemia. It is apparent from this study that even relatively small lesions in jejunum and ileum can be recognized by careful roentgen examination, and frequently their nature may be predicted prior to surgical exploration. The routine small intestinal study following the ingestion of barium is the method of choice for roentgen examination of jejunum and ileum. However, when this does not reveal the source of bleeding, judicious use of intestinal intubation and injection of barium through the tube will demonstrate lesions in an additional number of patients.

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DISCUSSION

DR. FRED J. HODGES, Ann Arbor, Mich. This account of the detection of lesions of the small intestine is impressive and indicative of what may be expected when serious roentgen exploration of this portion of the alimentary tract is carried out. We have seen this afternoon a splendid project carried out with excellent technique.

The lesions discovered by the surgeons and the pathologists in the 72 patients with proved abnormality were about evenly divided, you will remember, between malignant neoplasms and benign lesions. It is a point of some interest that, of the 51 patients adequately examined by roentgen ray, detection resulted in a relatively high proportion, a fact which recommends active search of the small intestine when otherwise unexplained bleeding is the presenting symptom.

I was particularly interested that the more or less standard method of small intestinal examination was so highly productive and that the more particularized methods of examination produced helpful findings so infrequently. It

would be most interesting to surgeons, internists and radiologists to learn in what percentage of situations in which bleeding could not be otherwise explained the authors have been able to detect small intestinal lesions. I mention that because, from our viewpoint, that is a consideration of prime practical importance.

Dr. Collins pointed out very nicely that there are certain patients with annoying bleeding whose lesions cannot be detected by other methods of approach. I wish she might have added from her experience how often this method has been able to provide sound explanation for a hitherto unrecognized source of alimentary bleeding.

Dr. Collins (closing). I am sorry that I cannot answer Dr. Hodges' question. Initially, we had hoped to attack the problem from that aspect but we found that we could not do that satisfactorily in retrospect since we had no listing of all the patients who had bled. We have now set up a plan of listing all of the patients that come in with bleeding and we hope that in a year or two we will have the answer to that question.

REPORT OF A CASE OF EOSINOPHILIC GRANULOMA OF BONE WITH ROENTGENOGRAPHIC DEMONSTRATION OF A SEQUESTRUM

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THE term "eosinophilic granuloma of bone" was suggested by Lichtenstein and Jaffe[4] in 1940 to denote a particular granulomatous lesion of bone which did not fit any existing classification. Several osseous defects with similar characteristics were described earlier by Schairer[6] in 1938, by Finzi[1] in 1929, and by Otani and Ehrlich[5] in 1940. Schairer considered the lesion to be an osteomyelitis with eosinophilic reaction; Finzi described it as a myeloma with prevalence of eosinophilic cells; Otani and Ehrlich believed it to be a benign granuloma of bone which simulated primary neoplasm.

Since the first suggestion of eosinophilic granuloma as a new and distinct disease entity, the term has been almost universally accepted. From 1940 to the present year, there have been approximately 200 cases of eosinophilic granuloma reported in the combined American and foreign literature.

ETIOLOGY

The cause of the disease is unknown. Otani and Ehrlich[5] concluded from their series of cases that trauma may play some role in initiating the lesion. The general consensus at this time has been expressed by Green and Farber[2] when they conclude that trauma merely precipitates symptoms in an already existing lesion and is not an etiologic factor. Virus and bacteria have been suspected as causative agents but neither has ever been recovered from a lesion of eosinophilic granuloma. The majority of authors at the present time concur that eosinophilic granuloma is closely related to Letterer-Siewe and Hand-Schüller-Christian disease. They feel that all three are different stages of the same basic disorder of the reticuloendothelial system.

PATHOLOGY

The lesion grossly appears reddish-brown when hemorrhage predominates and gray or yellow when necrosis and fatty infiltration are present without hemorrhage. Usually, cancellous and cortical bone are destroyed and often soft tissue is invaded. The wall of the cyst is made up of periosteum or a thin shell of reactive new bone.

Hamilton *et al.*[3] reported one case in which several small bone fragments were found at operation.

Microscopically, the basic component of the lesion is the histiocyte. This cell may have a foamy appearance containing lipoid deposits, phagocytosed blood pigment and eosinophilic granules. These cells are usually arranged in a reticular pattern. In addition to this basic cell, there are numerous leukocytes, mainly eosinophils; multinucleated giant cells which are most frequently found in hemorrhagic areas along the borders of bone trabeculae; and products of destruction such as necrotic debris and blood pigments. Products of repair such as fibrous reaction and callus bone are seen in some of the granulomas.

Otani and Ehrlich[5] reported one case where partially destroyed fragments of bone were observed. Green and Farber[2] reported finding particles of intact bone within large mononuclear phagocytes.

ROENTGENOGRAPHIC APPEARANCE

Because of the marked osteolytic property of eosinophilic granuloma, it char-

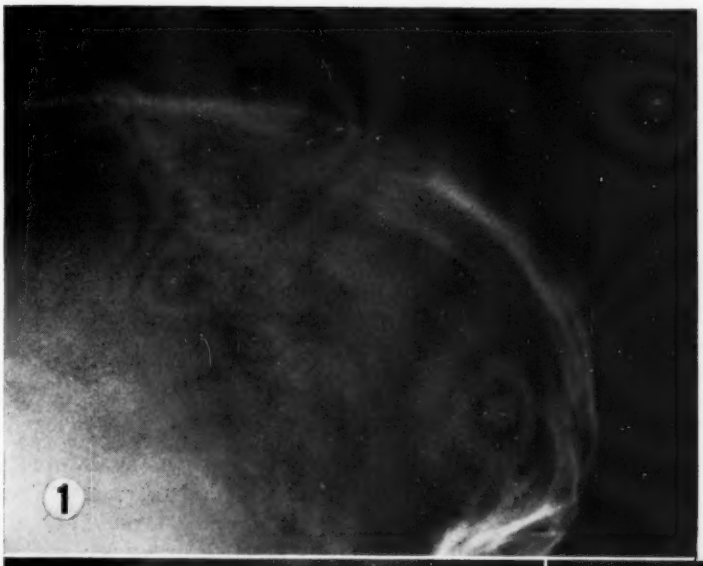

FIG. 1, 2 and 3. Roentgenograms show a sharply circumscribed defect involving both tables of the skull with no surrounding bone reaction. There is a central area of increased density with ill defined margins representing a large sequestrum.

acteristically produces round or oval punched-out, translucent areas, sharply demarcated from normal bone. Occasionally, there is a thin line of increased density surrounding the translucent area. Bone reaction in general is minimal. In long bones, the granuloma starts centrally and destroys cortex as it expands. Occasionally, expansion of the diaphysis with minimal periosteal reaction is noted. Frequently the lesion will break through the periosteum and invade soft tissue. In these cases, pathological fracture through the tumor area is common. After careful review of the literature, we were unable to find any case report of eosinophilic granuloma which showed roentgenographic evidence of sequestration.

Eosinophilic granuloma may be single or multiple. Approximately 30 per cent of

the cases will show multiple foci. Most common sites of involvement are skull, ribs, pelvis, spine and long bones. Foci, however, have been described in every bone except the small bones of the hands and feet. In the long bones, epiphysis, metaphysis, and diaphysis are involved with equal frequency.

Differential diagnosis of the solitary form of eosinophilic granuloma should include osteomyelitis, tuberculosis, syphilis, solitary bone cyst, fibrous dysplasia and giant cell tumor. When multiple lesions are found, neuroblastoma, lymphoma and osteitis fibrosa cystica must be considered.

REPORT OF A CASE

The patient, a white male, aged eighteen, entered this hospital on January 29, 1951, with a history of a blow to the occiput from a rifle butt, four weeks prior to admission. There was no laceration but, within a few days after the injury, the patient noted a swollen tender area over the left frontal bone. He also complained of frequent headaches since the time of injury. No neurological symptoms were noted. Physical examination revealed a moderate sized swelling, approximately 5 cm. in diameter, over the left frontal region. The swelling appeared to be inflammatory in nature, exhibiting redness, tenderness, increased local heat and some fluctuation. The scalp was intact. Otherwise, physical examination revealed nothing significant.

Laboratory tests were all within normal limits, including Bence-Jones protein and serum calcium.

Roentgenograms of the skull (Fig. 1, 2, and 3) revealed a circular area of increased radiolucency, approximately 2.5 cm. in diameter, involving both tables of the frontal bone. The margins of this defect were sharply defined with no evidence of adjacent osseous reaction. There was no roentgenographic evidence of soft tissue tumor. In the center of the defect was a sequestrum, measuring approximately 2 cm. in diameter, with ill-defined, moth-eaten margins. Density of the sequestrum was only slightly greater than that of normal surrounding bone.

Eosinophilic granuloma was not considered in the differential diagnosis because of this sequestrum. The roentgenographic impression was "localized osteomyelitis with sequestration, possibly tuberculous." Skeletal survey failed to reveal any additional foci of involvement.

Surgical exploration was performed on February 9, 1951. A focus of destroyed frontal bone, measuring 2 cm. in diameter, was noted and a grayish granulomatous tissue was seen in the area of involvement. It was adherent both to the galea and to the dura, and contained a large fragment of spongy necrotic bone.

Two separate specimens were submitted for pathological examinations. Specimen A consisted of a mass of soft, grayish tissue. Specimen B was a pancake-shaped piece of moth-eaten bone.

Microscopically the grayish tissue showed a predominance of foamy histiocytes; a few fibrocytes but little stroma; many areas of dense collections of eosinophils; and an occasional multinucleated giant cell, Touton variety. The second specimen revealed necrotic dead bone which presented empty lacunae and osteoclastic activity.

These specimens were reviewed by the Armed Forces Institute of Pathology and the diagnosis of eosinophilic granuloma of bone was confirmed.

SUMMARY

A case of eosinophilic granuloma of bone is presented. We believe this case to be important because of the unusual roentgenographic demonstration of sequestration within the lesion.

Sequestra of microscopic size within foci of eosinophilic granuloma have been described by several authors. One case has been reported in which small sequestra were seen grossly at the time of operation. However, of some 200 cases of eosinophilic granuloma reported in the literature, no reference is made to roentgenographic demonstration of sequestration.

CONCLUSIONS

1. Sequestration does occur in lesions of eosinophilic granuloma of bone.
2. The sequestration can be demonstrated roentgenographically.
3. Eosinophilic granuloma of bone should not be excluded from a differential

diagnosis because of the presence of a sequestrum.

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A VARIATION OF DIAPHYSEAL DEVELOPMENT WHICH SIMULATES THE ROENTGEN APPEARANCE OF PRIMARY NEOPLASMS OF BONE*

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NOWHERE in medicine is the borderline between the normal and the abnormal so ill defined as in the roentgenological study of the bony skeleton, and yet a knowledge of normal variations in structure and development is essential to the diagnosis of early pathological processes. Many of these developmental variations are so well known that they are seldom mistaken for abnormalities by the trained observer, while others are less widely known and recognized. Since Köhler's classical publication, *Röntgenology: The Borderlands of the Normal and the Early Pathological in the Skiagram,* is now almost unobtainable, the best combined source of information on developmental variations with which we are familiar is Caffey's *Pediatric X-ray Diagnosis.*

The purpose of this paper is to present a developmental variation of the skeletal system, which it is believed has not been previously described. The importance of this variation rests chiefly on the closeness with which it simulates or duplicates the appearance of a malignant bone neoplasm. Since the region of the knee is the site of numerous irregularities of development (*i.e.*, irregular calcification of the tibial tubercle; irregular calcification of the patella; irregular mineralization of the distal femoral epiphyses, especially the medial portion; physiological cortical defects; and accessory ossicles), it is not remarkable that the site of the lesion being described is the popliteal surface of the distal portion of the femoral diaphysis. The abnormality has been located superior to the epiphyseal line in all cases thus far observed and therefore is a variation in diaphyseal rather than epiphyseal development. The essential feature is an irregularity of mineralization in the shaft portion of the cortex of the popliteal surface of the femur, which is best seen in lateral projection.

The circumstances under which Cases I and II were encountered emphasizes the importance of recognizing the true nature of this condition. The great frequency of bone neoplasms in the region of the knee further emphasizes the importance of recognizing this developmental variation.

CASE I. The patient was an athletic white male, aged fourteen, who first sought medical advice on November 1, 1948, because of swelling and pain in the right knee, of three or four days' duration. The pain was accentuated by weight bearing or flexion. About five days before the first observation, there had been a slight injury which hardly seemed adequate to account for the clinical finding.

The entire physical examination revealed no significant abnormality except for the right lower extremity. The local temperature of the knee was elevated and palpation indicated the

FIG. 1. Lateral roentgenogram of Case I. Note posterior surface of femur above the epiphyseal line.

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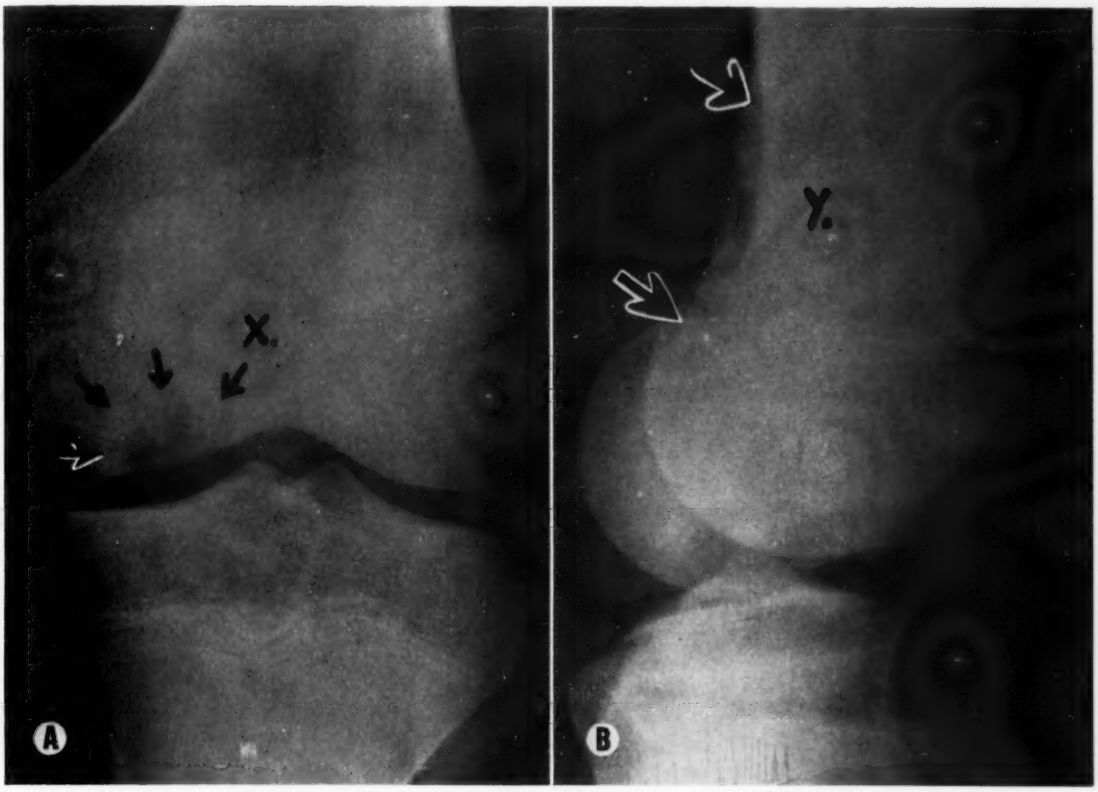

Fig. 2. Anteroposterior (*A*) and lateral (*B*) roentgenograms of Case II showing osteochondritis dissecans, *X*, in addition to the diaphyseal variation, *Y*.

presence of a moderate amount of fluid within the joint.

Roentgen examination of the knee (Fig. 1) in anteroposterior, lateral, and oblique projections showed a serrated margin to the posteromedial aspect of the femur. In stereoscopic views, this appeared to lie along the medial supracondylar line. Some of this region appeared to exhibit bone destruction although superiorly there was evidence of bone production and a soft tissue shadow having the appearance of a mass lying adjacent to this. This area lay just above the epiphyseal line on the distal femur.

These findings were interpreted as indicative of a malignant bone neoplasm, probably an osteogenic sarcoma. On this basis, the parents were urged to give their consent to biopsy to be followed by amputation if the roentgenographic diagnosis was substantiated pathologically. They steadfastly refused any form of surgical procedure in spite of great effort by the attending physicians.

Physiotherapeutic management was begun with such good results that the patient became asymptomatic and has remained free of complaint. During the intervening fourteen months, he has engaged in such sports as basketball and baseball without difficulty.

Case II. (Seen through the courtesy of Dr. E. H. Balch of Seymour, Texas.) The patient was a white male, aged fourteen, who first sought medical advice about April 1, 1949, with a complaint of pain and swelling of the right knee. Physical examination revealed no abnormality except swelling and tenderness of the right knee.

Roentgenograms of the knee (Fig. 2) in anteroposterior and lateral projections revealed an irregular area on the posterior aspect of the femur which began at the distal epiphyseal line and extended upward for a distance of 3 cm. The appearance was almost identical with Case I. An additional finding, however, was an area on the articular surface of the lateral aspect of the medial condyle of the femur which had the appearance of osteochondritis dissecans. Having had the experience with Case I, the roentgen

impression on Case II was "osteochondritis dissecans, plus a developmental variation of the lower femoral shaft."

Clinical follow-up eight months later revealed some locking of the joint, with intermittent episodes of mild pain, but there was no evidence to suggest the presence of a neoplasm. It has not been possible to make follow-up roentgenograms.

CASE III. The patient was a white female, aged fourteen, who was first seen at Presbyterian Hospital, New York City, on March 1, 1945, because of an injury to her knee suffered during play the same afternoon.

Physical examination revealed no significant evidence of disease.

Roentgenographic examination of the knee (Fig. 3) was carried out. The interpretation by Dr. Ross Golden described an irregularity on the posteromedial aspect of the distal femur above the epiphyseal line. The report reads in part as follows, "The appearance of the lower half of this area must be considered as very suggestive of a small malignant tumor, possibly an osteogenic sarcoma, but it also could be a Ewing tumor. It seems possible that the upper part of the elevation might represent an old completely ossified subperiosteal hematoma."

FIG. 3. Lateral roentgenogram of Case III showing a diaphyseal irregularity.

Surgical exploration was done by Dr. Fred Smith. When the area was exposed, there was definite evidence of a periosteal thickening. The bone showed slight softening, but there was no evidence of tumor.

Pathological examination by Dr. A. P. Stout revealed some degree of calcification on the periosteal and extraperiosteal aponeurotic tissue. There was no evidence of tumor, and he offered no suggestion as to the etiology of the process.

Later roentgen examination showed that the biopsy had included the entire abnormal area described on the first roentgenographic examination. During a four year follow-up period, the patient experienced no further symptoms referable to the knee and has remained in good health.

Comment. When Case I was seen, the roentgen findings were so typical of the usual appearance of an osteogenic sarcoma that the diagnosis was made with little hesitation.

The prompt and lasting response of Case I to conservative management occasioned a much more cautious interpretation when Case II was seen six months later. Conservatism seemed further justified by the coexistence of an osteochondritis dissecans, which seems to supply adequate explanation for symptoms and findings. In view of the experience in Case I, plus this additional lesion, conservative management was advised and subsequently carried out.

The observation of these 2 cases in a six month period led the author to seek the same findings in asymptomatic individuals in this same (fourteen year old) age group.

Lateral views of 26 knees on fifteen year old white asymptomatic males revealed 4 examples of changes similar to those noted in Cases I and II. One of these was as marked as the reported cases while the other 3 were of lesser magnitude.

The author is particularly grateful to the Radiological Service of Presbyterian Hospital for Case III, since this is the only case for which pathological proof of benignancy is available. The four year follow-

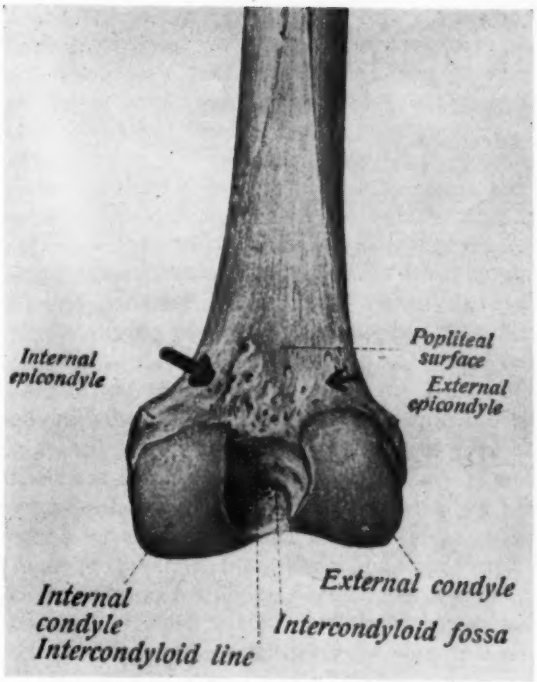

Fig. 4. Posterior view of lower femur showing numerous perforating blood vessels on the popliteal surface. (From Sobotta and McMurrich. Atlas of Anatomy. W. B. Saunders Co., Philadelphia, 1906, p. 98.)

up is also the longest yet available on this lesion.

Our present state of information provides no proved explanation for this variation in development. It has been suggested by Caffey* that the appearance may be related to the presence of numerous small perforating blood vessels (Fig. 4) in this part of the popliteal surface of the femur. The rather marked variation in the size and number of these vessels in different individuals may explain why this abnormality being described occurs in only a limited percentage of the population. This, of course, is an unproved, though plausible explanation.

* Caffey, J. Personal communication.

SUMMARY

1. The importance of the variation of bone development being presented rests upon the similarity of the roentgen appearance of this lesion and osteogenic sarcoma.

2. This variation is a variant of diaphyseal rather than epiphyseal development.

3. The observed age of incidence is fourteen to fifteen years.

4. The typical roentgen finding is a serrated irregularity of the cortical margin of the popliteal surfaceof the femur affecting the 3 to 5 centimeters immediately proximal to the distal epiphyseal line. There is a loss of definition of the cortical margins. This bony change has usually been associated with a soft tissue shadow simulating an adjacent tumefaction.

5. A negative biopsy of one case, clinical follow-up of two cases, and the demonstration of similar roentgen findings in asymptomatic individuals of the same age group are submitted as evidence that this is a developmental variation, rather than a neoplasm.

6. The irregularity may be produced on the lateral roentgenogram by the superimposition of the foramina of the perforating nutrient arteries in this right angle view. If this is true, the presence or absence of the roentgen findings being described depends upon the number and distribution of these perforating nutrient arteries.

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INTRAUTERINE PERFORATION OF MECKEL'S DIVERTICULUM*

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A COMMON postmortem finding is the presence of a Meckel's diverticulum which is found in about 2 per cent of all bodies examined.[2] This congenital outpouching of the ileum is a remnant of the omphalomesenteric duct, and, according to Meckel,[5] is a result of an arrest in the development of the small intestine during the period when it is still connected with the umbilical vesicle. The diverticulum is usually found 30 to 90 cm. proximal to the ileocecal junction and projects from the free border of the ileum. Although rarely of clinical interest, symptoms may occur in a small percentage of individuals who develop complications. Among the complications described are ulceration and perforation of the diverticulum, volvulus, gangrene, and intestinal obstruction from incarceration of a loop of intestine due to adhesions or formation of a hernial sac. Within the diverticulum, heterotopic gastric, pancreatic, or duodenal mucosa may be found. The gastric mucosa may secrete gastric juice and cause a typical peptic ulcer.[6] Hemorrhage or inflammatory changes in an ulcer constitute the most frequent causes for symptoms of Meckel's diverticulum. Pfahler[8] observed one of the first cases roentgenographically in 1923 and reported it in 1934.

The case to be reported is of interest, primarily, because one of the complications of Meckel's diverticulum, ulceration and perforation, occurred during gestation and caused the appearance of roentgen findings which are commonly associated with meconium ileus. Only subsequent postmortem examination nine hours after birth disclosed the true nature of the roentgen findings.

REPORT OF CASE

Family history was noncontributory. The pregnancy was uneventful except for the presence of hydroamnion. This newborn female infant was delivered in the eighth month of gestation. The delivery was difficult because of abdominal distention of the infant, but forceps were not used. Aside from the difficulty in the delivery of the child due to the distended abdomen, the birth was uneventful and the child was in fairly good condition. However, shortly after the birth the infant was observed to be dyspneic. The abdominal veins appeared markedly distended. A good deal of mucus was discharged from the nose and mouth, and because of poor color and labored respiration, the infant was placed in an oxygen tent with continuous administration of oxygen. An emergency roentgenogram of the chest and abdomen was made (Fig. 1). The abdomen showed considerable bulging of the flanks with absence of the properitoneal fat lines bilaterally. At the lateral borders of the abdomen and also at the right upper quadrant, irregular mottled calcifications were observed which were quite extensive in character. A moderate degree of gaseous distention of the stomach and small intestine was observed. However, no gas was present in the colon. The roentgen diagnosis was "meconium ileus with peritonitis, and intestinal obstruction probably localized at the ileocecal junction." The infant became progressively more dyspneic and weaker and nine hours after delivery died. At death the infant had a pseudomenstruation and passed a large mucus plug from the rectum which was tipped with meconium. There had been no previous bowel movement.

At postmortem examination* the pertinent findings were limited to the abdominal cavity. The gross specimen of the small intestine showed a diverticulum measuring 3.5 cm. in

* Pathological studies performed by Dr. Ancel Blaustein, Pathologist, Bayonne General Hospital.

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length and 2.5 cm. in diameter present in the ileum approximately 3 cm. from the ileocecal junction. The diverticulum was ruptured and the gap covered over by thickened peritoneum which itself was the site of a marked inflammatory reaction and had a thick greenish-brown fibrinopurulent exudate on its surface. The walls of the diverticulum were thin, necrotic and also covered with this patchy exudate. The cut surface of the intestine revealed a hemorrhagic, necrotic wall. The mucosa in the diverticulum was largely sloughed and the material present in the luman of the intestine was foul smelling.

Microscopically (Fig. 2 and 3), sections taken from the ruptured diverticulum showed hemorrhagic necrosis with marked leukocytic infiltration extending into portions of what appeared to be normal ileum. There was no recognizable gastric mucosa present within the diverticulum nor any pancreatic tissue present either on the mucosal surface or within the submucosa of the intestine. On the serosal surface there was both an exudative and productive reaction consisting

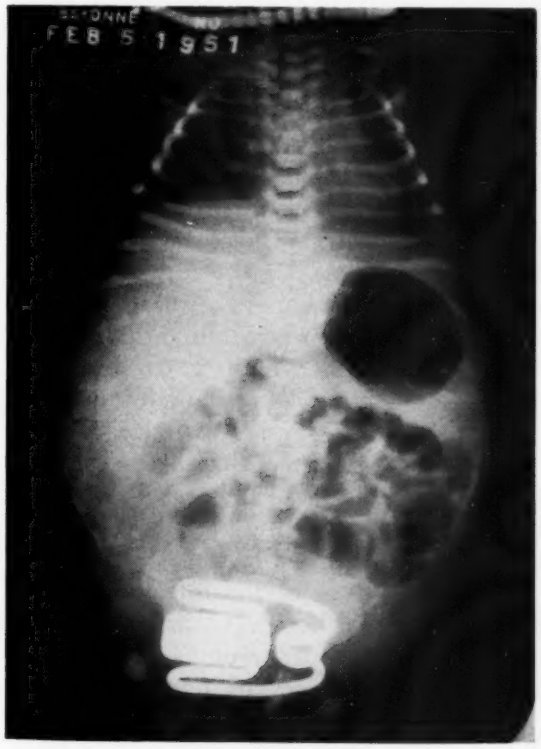

Fig. 1. Flat film examination of the abdomen and chest reveals extensive irregular calcifications within the abdomen. The loops of the small intestine and stomach are distended with gas.

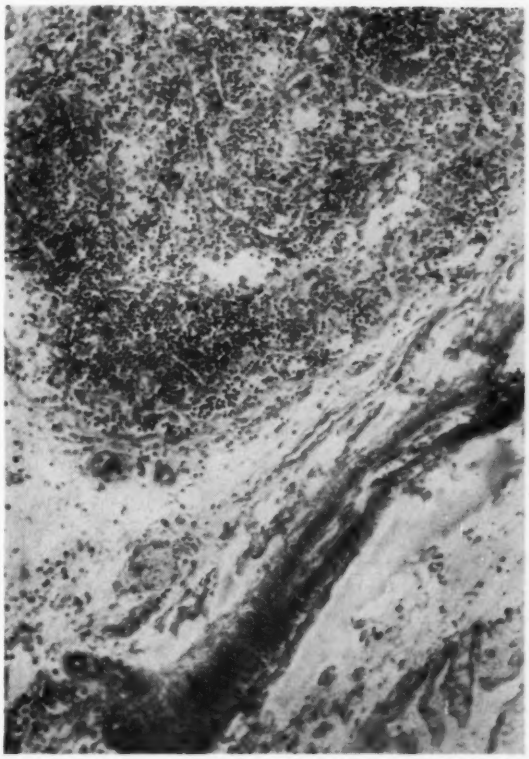

Fig. 2. Marked exudative reaction at the site of perforation extending from the submucosa through the serosa. The reaction consists of polymorphonuclear leukocytes, lymphocytes, and macrophages (100×).

of fibrosis, polymorphonuclear leukocytes, mononuclears and a large number of macrophages filled with old blood pigment, and in the wall large numbers of macrophages were present which appeared to contain material resembling meconium. There was also marked fibrosis extending into the peritoneum which was adherent to the serosa of the intestine. There were calcific deposits scattered throughout the fibrous tissue.

The peritoneum was markedly thickened and grossly appeared covered with a thin, dry, brown, sand-like granular deposit on the parietal surface. On the visceral surface, there were some patchy, green, fibrinous deposits intermingled with irregular calcific deposits. The peritoneum was adherent to the ileum.

Microscopically, the peritoneum showed both exudative and productive responses with marked infiltration of polymorphonuclear leukocytes, monocytes and macrophages as well as a fibrinous reaction. Patchy calcific deposits and macrophages laden with greenish-brown

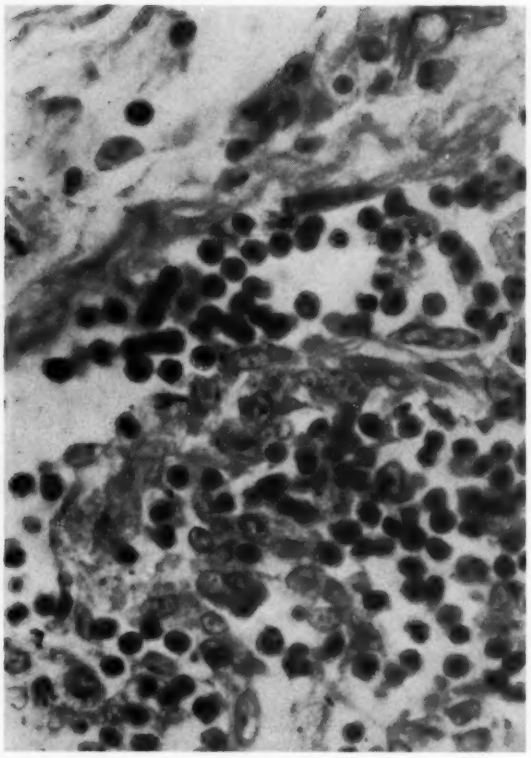

FIG. 3. High power photomicrograph of the same field showing the macrophages filled with cellular debris, as well as the other cellular elements representing a severe inflammatory reaction (400×).

pigment were found close to the visceral surface.

The pathological diagnosis was intrauterine perforation of Meckel's diverticulum with meconium peritonitis.

DISCUSSION

One of the most interesting features of Meckel's diverticulum is the frequent presence of heterotopic gastric, pancreatic or duodenal mucosa. Aberrant gastric mucosa, capable of secreting a potent digestive juice accounts for the occurrence of typical peptic ulcer in these pouches.[1] Ordinarily, the ulcer is found in the intestinal mucosa bordering on the heterotopic gastric mucosa. All of the symptoms and complications of duodenal ulcer may be found in association with ulcer of Meckel's diverticulum. The patients are most commonly admitted to the hospital because of perforation or peritonitis, or both. In other cases anemia, melena, or persistent occult blood in the stool are the only findings of importance. In some patients, pain in the umbilical region or in the right lower quadrant may be present intermittently for an indefinite period before the onset of more serious complications. In very young individuals presenting complaints of this type, the possibility of a Meckel's diverticulum should be considered. The final diagnosis frequently is dependent upon an exploratory operation when peritonitis or obstruction has not resulted since it is extremely difficult to make the diagnosis on routine roentgenographic studies. Hemorrhage from erosion, ulceration, or inflammatory changes in the diverticulum constitutes one of the most common symptoms. Periodic attacks of severe internal bleeding in the absence of other symptoms in childhood or early adult life should suggest this possibility if no other cause can be found for the bleeding. A familial history of other congenital anomalies may be helpful.[3]

Tumors have been reported within these diverticula. Horsley and Keasby[4] compiled 20 cases from the literature in 1939 including 5 fibrosarcomas, 5 leiomyosarcomas, 5 carcinomas, and 5 carcinoid tumors. Adenoma has also been reported in this situation.

In the case reported, it is probable that heterotopic gastric mucosa was present, but was destroyed by the intense inflammatory reaction. This in turn would suggest the interesting possibility that, under certain conditions, fetal gastrointestinal glands may function, although normally these glands do not start to function until after birth. Since no other case of intrauterine perforation of a Meckel's diverticulum was found in the literature, evaluation of this factor is difficult.

The appearance of the intra-abdominal calcifications, although striking, are not unusual or pathognomonic. Neuhauser[7] has reported 3 cases of fetal meconium peritonitis in whom symptoms and signs shortly after birth made roentgen studies advisable. Here, too, scattered intra-abdominal calcifications were noted indicating perforation and peritonitis during gestation. He emphasizes the difficulty in visual-

izing the site of perforation and postulates that the perforations occurred some time before delivery but after the fifth month of intrauterine life. His 3 cases at autopsy or operation revealed the presence of meconium in the peritoneal cavity.

No free meconium was found in the peritoneal cavity of our case, but the appearance of the peritoneum was the same as is found in meconium ileus, with perforation and obstruction.

CONCLUSION

A case of intrauterine perforation of a Meckel's diverticulum is described. No similar case has been found in a review of the world literature. The similarity of the roentgen findings to those of meconium ileus with perforation is stressed.

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FEMALE PSEUDOHERMAPHRODITISM

REPORT OF A CASE PROVED ROENTGENOLOGICALLY*

By BERTRAM LEVIN, M.D., ALWIN C. RAMBAR, M.D., *and* IRVING J. SHAPIRO, M.D.

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HETEROSEXUALITY has been recognized as an entity worthy of special attention since biblical times. This developmental anomaly is of interest not only because it represents a freakish condition, a curio, but also because of its sociological, psychological, endocrinological, surgical, and moral implications. With better understanding of adrenal physiology and pathology and with improvement in surgical techniques, pseudohermaphroditism has become a problem of even greater importance because reparative treatment is now available for selected cases.[4]

Heterosexuality, further classified, includes true hermaphroditism, male pseudohermaphroditism, and female pseudohermaphroditism.

True hermaphroditism is probably the rarest form of heterosexuality; the term is applied to individuals having gonads of both sexes. There may be one ovary and one testis, ovotestes, or an ovotestis with an ovary or testis. In discussing the etiology, Wilkins states that "many workers assume that such mixtures of the sex character of the gonads must be due to abnormal combinations or mosaics of the genetic pattern . . . [But it is] unwise to exclude the possibility that non-genetic or hormonal factors might be responsible for the development of ovotestes in the human."[14]

Male pseudohermaphrodites are individuals having testes but also female external genitalia, genital ducts, or both. As yet too little is known about this condition to speculate on its cause.

Female pseudohermaphroditism due to congenital hyperplasia of the adrenal is the commonest cause of heterosexuality. These individuals have ovaries but male development of some of the accessory sex organs. Wilkins states that all the pseudohermaphroditic patients with purely ovarian gonads "presented evidence of excessive androgen production throughout life, indicated during early childhood by precocious masculine sexual development, advanced growth and osseous development and increased 17-ketosteroid excretion. Such patients generally have shown at autopsy or biopsy evidence of adrenal hyperplasia or overactivity."[14]

To determine the nature of the internal sex organs surgical exploration and biopsy are usually necessary.

Heterosexuality often presents a diagnostic problem to the pediatrician, internist, and surgeon; the radiologist may thus be consulted. Generally the radiologist can be of assistance in diagnosis only in determining whether or not there is precocious skeletal development, adrenal tumor, or both. If a urogenital sinus is present, films taken after the injection of a contrast medium may show a vaginal pouch and its communication with the urinary bladder. Thus, roentgenologic methods are usually of aid only in providing circumstantial evidence for or against the clinical diagnosis of heterosexuality.

The purpose of this paper is to report a case of female pseudohermaphroditism conclusively proved by roentgenographic methods. After careful perusal of the literature, we believe this to be the first case to be proved in the manner described below.

CASE REPORT

S. S., a five year old white "male," was admitted to Sarah Morris Hospital for Children,

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Michael Reese Hospital, for urologic and endocrinologic study.

The child had been a full term infant following an uneventful pregnancy and uncomplicated delivery. At birth he was noted to have some genital abnormality; a hypospadias was reported to be present.

The child's development progressed normally until a few months before admission. At this time there was noted appearance of abundant coarse black pubic and axillary hair. An adrenal tumor was suspected and the patient was referred elsewhere for an intravenous urogram. This showed no evidence of adrenal tumor or other abnormality (Fig. 1).

The patient was then admitted to Sarah Morris Hospital for further study.

Physical examination revealed a well developed muscular boy, 49 inches in height, somewhat taller than expected for his chronological age. According to the chart of Burgess[2] showing height of American boys and girls at various ages the patient is above the 99 percentile. He weighed 51¾ pounds. His weight in relationship to his height was normal according to the tables of Engelbach.[3] Examination of head, neck, heart, and lungs showed no abnormality. Abundant black, coarse pubic hair was present. There was beginning axillary hair growth and several hairs over the sternum.

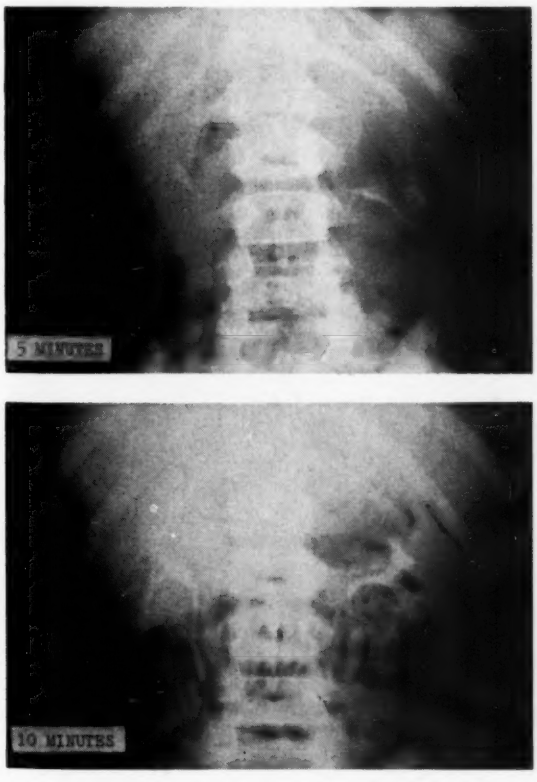

Fig. 1. Excretion urograms (five and ten minute films) showing normal position of kidneys and calyces.

Fig. 2. Left wrist. The precocious skeletal development is evident.

Examination of the genitalia revealed a penis-like structure with a urethral opening below it. There were well developed labia or unjoined scrotal folds. No gonads were palpable.

The clinical impression was that this represented a case of androgenic precocious puberty with congenital abnormality of the external genitalia. An adrenal functional tumor was considered likely.

Blood studies and routine urinalysis showed no abnormality. Twenty-four hour pregnandiol excretion was 99 mg. The normal value for the method of determination used is less than 1 mg. 17-ketosteroid excretion was not determined.

Roentgen Findings. Examination of the skele-

tal system revealed precocious osseous maturation, the bone age being that of about an eleven year old (Fig. 2).

Air was injected into the left perirenal space. Films taken with the patient upright outlined a normal sized left kidney. The suprarenal gland did not appear to be enlarged. Above the kidney and lateral to the suprarenal gland there was a large soft tissue mass which was continuous with the shadow of the spleen (Fig. 3). It could not be determined whether this represented a spleen of unusual configuration or whether it represented some form of neoplasm, extra-adrenal.

Three days later a pneumoperitoneum was produced under general anesthesia by injecting 500 cc. of oxygen into the peritoneal cavity by the transabdominal route. Films taken in the upright position showed that the previously described left upper quadrant mass was intraperitoneal and continuous with the spleen, probably representing an unusually prominent upper pole of spleen (Fig. 4).

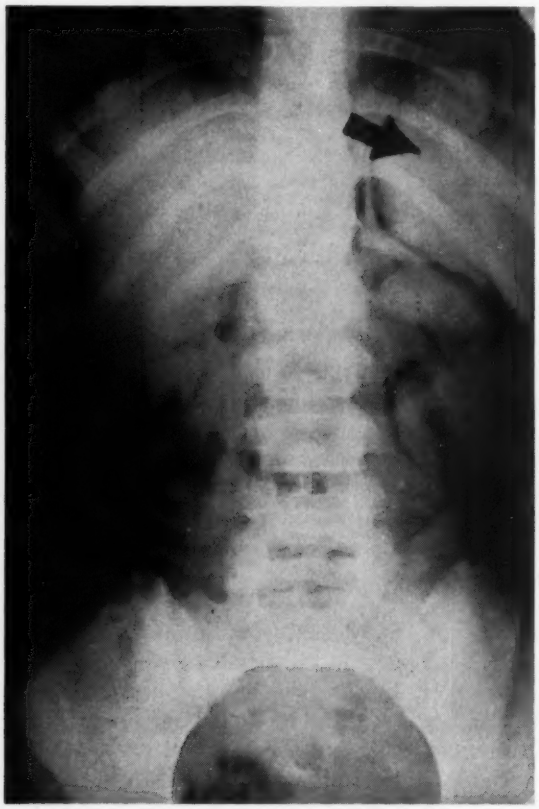

FIG. 3. Left perirenal air insufflation. Arrow points to soft tissue mass of indefinite location and nature.

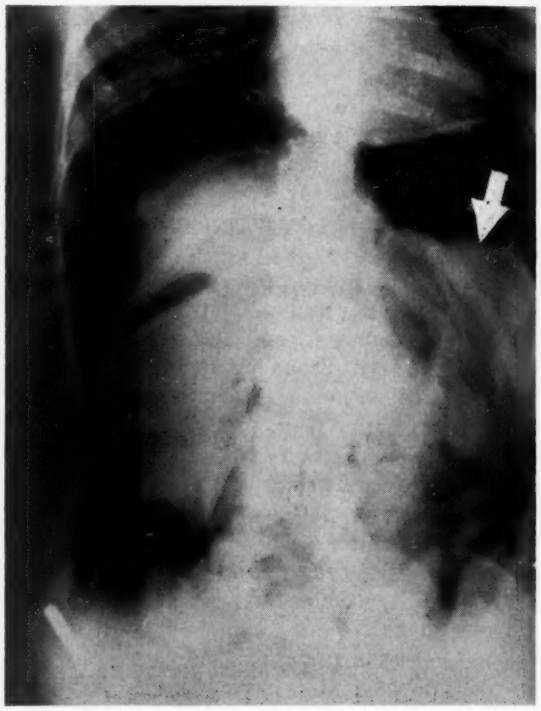

FIG. 4. Pneumoperitoneum. Film taken in upright position. Mass (arrow) noted in Figure 3 is seen to lie within the peritoneal cavity since it is displaced by the gas.

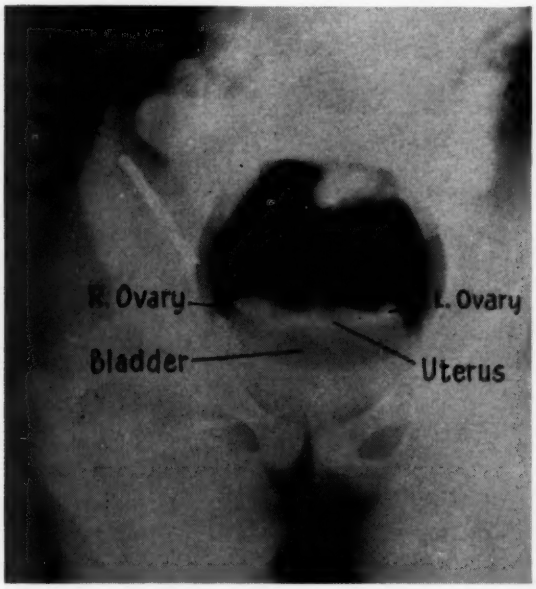

FIG. 5. Pneumoperitoneum. Film taken in modified knee-chest position. Infantile uterus and ovaries are well visualized.

Films taken with the patient in a modified knee-chest position revealed the outlines of an infantile uterus and ovaries. These were normal in size, shape and position for a female of this age (Fig. 5).

The following day the patient underwent exploratory laparotomy. A left subcostal incision was made and the peritoneum opened. A small uterus, tubes, and adnexa were palpated. Palpation above the right kidney seemed to reveal an enlarged suprarenal gland, but it was difficult to be sure of this. The posterior peritoneum was opened in the left lateral gutter and blunt dissection carried out above the kidney, exposing an enlarged left suprarenal gland. A piece was removed for section.

The patient made an uneventful recovery from the exploratory operation.

The suprarenal biopsy specimen was reported (Dr. O. Saphir) as showing "hyperplasia of the zona reticularis and recent hemorrhage and edema."

DEVELOPMENT OF PNEUMOROENTGENOGRAPHY

In 1912 Jacobaeus[5] was the first to call attention to the great advantage of combining pneumoperitoneum and roentgen examination. In 1919 Orndoff[6] reported on the use of pneumoperitoneum combined with roentgenography as a diagnostic aid. Most of his 100 cases involved adhesions between the intestine and abdominal wall or had upper abdominal lesions. In this report he stated as follows: "The size, position, mobility, relative density, variations in density, contour, contents, and cavities of the abdominal viscera can be visualized and studied in a manner which opens to physicians entirely new possibilities."

In 1921 Van Zwaluwenburg and Peterson[13] demonstrated the practicability of pneumoroentgenography for gynecologic diagnosis. They used a modified Rubin patency technique for transuterine inflation and developed the technique of transabdominal puncture for cases where the tubes were obstructed or where the transuterine inflation was contraindicated. They also described a modified knee-chest position for roentgenography to better visualize the pelvic viscera.

In that same year (1921) Alvarez[1] was the first to advocate the use of carbon dioxide in pneumoperitoneum. This gas is absorbed much more rapidly than is air or oxygen, and consequently the patient suffers little, if any, distress due to tension. The CO_2 is usually almost completely absorbed in thirty minutes.

One year later, in 1922, Stein and Arens[11,12] adopted the method in this hospital and since then it has become a useful and often used procedure in the armamentarium of the gynecologists. They developed a self-retaining patency set and a special roentgenographic table which facilitates roentgenography with the patient in a modified knee-chest position and the Potter-Bucky diaphragm close to the abdominal wall. This table has been improved with the passing years; the latest design is a modified Sisk cystoscopic table.

The indications and technique for pneumoroentgenography in adults have been adequately described elsewhere.[10,11,12,13,14] Briefly, it consists of introducing approximately 1,000 to 1,500 cc. of air, oxygen, or CO_2 into the peritoneal cavity via the transuterine or transabdominal route. The latter is used when the transuterine route is unsuccessful or contraindicated. For transabdominal pneumoperitoneum the puncture site is first prepared with a suitable antiseptic. A No. 17 needle, $3\frac{1}{2}$ inches long with stilet is used with a quick thrust through the abdominal wall at about 1 inch below and to the left of the umbilicus. This is done without any previous anesthesia to the skin. When the needle is in place and connected to a suitable apparatus for measuring and controlling the flow of the gas and as the gas is flowing one can tell whether the needle point is in the peritoneal cavity by listening to the abdominal wall with a stethoscope. A characteristic sound of an inrush of air can be heard. By the amount of abdominal distention, one can usually determine whether or not there is a satisfactory amount of intraperitoneal gas; about 1,000

to 1,500 cc. will usually be required.

For children the technique is essentially the same. The age of the child will determine whether or not general anesthesia is necessary. The transabdominal route is used, and correspondingly smaller amounts of gas, depending on the size of the patient, are employed to produce pneumoperitoneum. Probably no less than 350 cc. should be used. This technique has been used successfully in this hospital to confirm a number of cases of ovarian agenesis or hypoplasia. As in the reported case, oxygen is usually used in young children, since the rapid absorption of CO_2 does not permit repeating the films, should such be required, without introducing more gas.

SUMMARY

A case of female pseudohermaphroditism has been presented. It was conclusively proved by roentgen methods and is believed by the authors to be the first such case in which the female pelvic structures were demonstrated roentgenographically after producing a pneumoperitoneum. The findings were confirmed by operation.

Brief discussions of heterosexuality and pelvic pneumoroentgenography have been presented.

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VOL. 69, NO. 6

DIRECTED BEAM THERAPY

II. MULTIPLE SMALL FIELD IRRADIATION OF THE PITUITARY GLAND, PITUITARY TUMORS AND OTHER INTRACRANIAL LESIONS

By B. V. A. LOW-BEER, N. E. SCOFIELD, E. T. FELDSTED, *and* R. F. BROWN

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THE general aim in treating a deep-seated tumor with roentgen rays is to insure that the maximum of absorbed energy be confined to and uniformly distributed within an estimated tumor volume. Ideally, all tissues outside the volume to be treated should absorb no radiant energy. This, however, is not possible with any technique of roentgen treatment. Therefore, a compromise in which all surrounding tissues receive a much smaller amount of radiant energy than does the tumor may be considered as a "practical ideal."

The conventional techniques of employing one or two portals of entrance for the radiation beam directed to a deep-seated tumor result in the accumulation of an equal or greater dose on the surface of the body as compared with the dose received at the focal depth. This high surface dose is the principal disadvantage of the one portal or two portal "cross fire" technique within the 200 to 400 kv. roentgen-ray energy ranges which are usually employed. The "practical ideal" can be most nearly achieved when the irradiation is done through multiple portals of entrance. Rotational irradiation provides the maximum in multiple portals. When multiple portals are used, all beams converge on the lesion, and the parts of the body through which each beam passes receive a dose which is smaller than the total dose delivered to the tumor. Within certain limits, the ratio between tumor dose and either surrounding tissue dose or skin dose is increased by increasing the number of treatment portals. Factors which influence this ratio are: (a) quality of radiation, (b) tube target-tumor distance, (c) diameter of the object, (d) density of tissue through which the roentgen-ray beam passes, and (e) the relation of the tumor to the body contour in the segment of the body to be irradiated.

Theoretically, the highest differential ratio can be attained if there is a spherical body within which there is concentrically located another smaller spherical mass to be irradiated. When one considers (Fig. 1) the small body v within the larger body V and one chooses a set of three mutually perpendicular coordinate axes intersecting

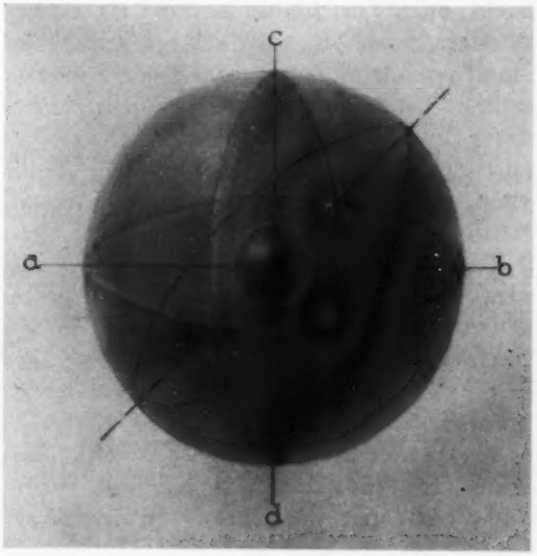

FIG. 1. This shows that when a cartesian coordinate system is chosen whose origin coincides with the center of the spheres, then when the axis of rotation of the spheres v and V is fixed, and a roentgen-ray beam of just sufficient size to enclose the small sphere is directed at the origin, the small phere will remain in the beam in any rotational position while different areas of the large sphere will be irradiated for each position. The greatest number of separate entrance fields on the surface for each axis may be obtained by directing the roentgen-ray beam perpendicularly to the axis of rotation.

* This study was made in 1949 and 1950 and was financially supported by the American Cancer Society's "Institutional Grant to the University of California School of Medicine, San Francisco."

at the center of the smaller body, the mass as a whole may be rotated around any of these axes without changing the location in space of the small body. If a roentgen-ray beam of just sufficient size to enclose the small body is directed at the center of rotation, the small body will remain in the beam in any rotational position. The greatest number of separate entrance fields on the surface for each axis may be obtained by directing the roentgen-ray beam perpendicular to the axis of rotation.*

In clinical radiation therapy, one does not deal with entirely spherical surfaces, but rather with segments of spheres or cylinders. Therefore, instead of approaching the volume to be treated through the entire surface of the hypothetical sphere, one is limited to the irradiation of a segment of the available surface. It is still possible, however, with a large number of small portals of entry, to deliver a greater amount of radiant energy to the tumor mass than is received by any of the tissues surrounding the tumor. This is possible even with roentgen rays generated by potentials in the 200 kv. range.

The accomplishment of the "practical ideal" has been the subject of study by several investigators. Dobbie was among the first to turn attention to the advantages of small field treatment and necessity of accurate beam direction.

The physical and geometrical principles underlying multiple field beam direction have been analyzed in detail by Mayneord.[9] Winternitz and Smithers[9] have described a technique whereby multiple small fields are arranged in a circle on the surface of the body equidistant from the center of the lesion. By means of a parallel arm beam director, roentgen-ray beams delivered through these fields pass through the body at an oblique angle to converge at the center of the lesion. Kaplan and Etkin[4] used a protractor to determine and maintain the angulation of the roentgen tube so that the central ray for each portal passed through the center of the lesion. Dobbie[2] described a method for making casts out of wax to fit the part of the body under treatment so that the alignment of the roentgen tube could be reproduced at each treatment. Dobbie used a protractor and an exit beam indicator to control beam direction.

Many other beam direction methods have been described in recent years.[3,7,8] The most widely accepted approach is the use of multiple narrow radiation beams converging on the tumor. All these *small field, multiple beam techniques* employ directional calipers on the parallelogram or arc principles.†

The present study was undertaken to provide a method of delivering to the pituitary gland a tissue dose of 8,000 to 10,000 roentgens in a period of from fifty to sixty-five days so that the skin dose on each portal would not exceed 3,000 to 4,000 r.

The reason for such intensive irradiation of the pituitary was an attempt to decrease the function of the pituitary gland and to affect tumor growth in patients with disseminated cancer.

Although no significant effect on pituitary function and on tumor growth could be demonstrated in the 5 patients so treated,[5] the method has proved of definite value in the treatment of pituitary tumors and also in the irradiation of other intracranial neoplasms.

Our special problem was to construct a beam alignment device which would enable us to irradiate the pituitary gland in accordance with the principles of a high differential dose ratio between the pituitary and the surrounding tissues.

The human head and the pituitary gland may be considered, for practical purposes, as spherical or oblate bodies concentrically located and can be described in terms of three mutually perpendicular coordinate

* If the body is of cylindrical form and the mass to be irradiated lies in the axis of the cylinder, then a roentgen-ray beam directed perpendicularly to the axis of rotation will give the greatest number of fields with the minimum depth from the surface to the focal volume.

† This study is concerned with multiple stationary fields and not with rotational irradiation.

axes. The point of intersection of the axes is the pivotal point of the geometrical figure (Fig. 2). If the axis *a–b* is vertical, then a horizontal plane can be laid through the axes *c–d* and *e–f*. If the center of a roentgen-ray beam of just sufficient size to enclose the pituitary is directed to pass through this horizontal plane and intersects *a–b*, any rotation of the head about the axis *a–b* will result in continuous irradiation of the pituitary and only intermittent irradiation of the skin and other tissues of the head within the limits of the roentgen-ray beam. By using this approach, the differential dose ratio between the skin and the pituitary gland depends mainly on: (1) the number of treatment fields, (2) the quality of radiation, and (3) size of the fields.

Based on the foregoing principles, a device was constructed which could be easily attached to the roentgen tube head and which insured reproducible position of the patient with accurate beam direction.

The device consists of two parts: (1) the roentgen-ray beam localizer and (2) a plaster cast skull cap fitted snugly to the patient's head. The roentgen-ray beam localizer is a frame made of 1 inch brass angle stock fashioned into two parallel interconnected arms projecting from the base plate of a roentgen tube head.

Since the arms of the frame are made parallel to and at a fixed distance from the central ray axis of the collimated roentgen-ray beam, they are a reference system located a fixed distance from the axis of the roentgen-ray beam. A wooden plate with a hole drilled through its center is affixed to and in the plane of the projecting arms. The hole serves to locate the axis of rotation. The position of the plate along the length of the projecting arms is adjusted so that the center of the "rotation hole" is 65 cm. from the anode of the roentgen tube.

The roentgen-ray beam is thus perpendicular to the vertical axis through the "rotation hole." The fixed known distance from the projecting arms of the frame to the center of the roentgen-ray beam is duplicated along the rotation axis, and thus the point at which the roentgen-ray beam intersects this axis is constant.

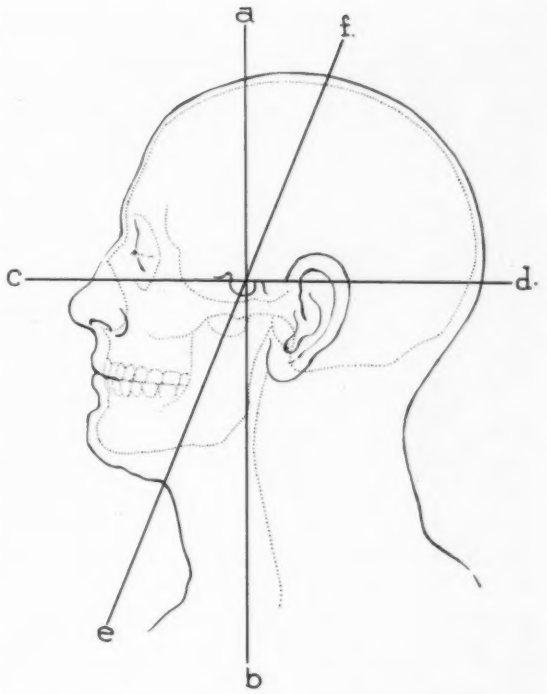

Fig. 2. The human head and the pituitary gland may be considered, for practical purposes, as spherical or oblate bodies concentrically located and can be represented by three mutually perpendicular coordinate axes. The point of intersection of the axes is the pivotal point of the geometrical figure.

A roentgen-ray beam perpendicular to the vertical rotation axis was chosen because by such arrangement the point of maximum dose coincides with the point of intersection of the roentgen-ray beams. It is somewhat simpler to set up and adjust the patient to the perpendicular radiation beam than to direct the beam at an angle toward the rotational axis.

The width of the beam is defined by two lead diaphragms attached to the projecting arms, one at the base plate of the roentgen tube head, the other at 50 cm. from the anode of the roentgen tube. The beam defining lead diaphragm is cut with a 2.2 cm. diameter circular hole, which provides a beam diameter of 2.8 cm. at

65 cm., the distance from the target to the center of the sella turcica.

For treatment of pituitary tumors, the hole defining the roentgen-ray beam is made larger to fit the tumor size. The largest beam size used was 3.8 cm., providing a beam diameter of 4.6 cm. at the center of the pituitary.

The beam size is constant regardless of the portal being used. The beam size of 2.8 cm. includes the normal sella turcica with a 2 to 3 mm. margin all around.

In order to irradiate the pituitary, the next step is to establish the craniocaudal rotational axis passing through the pituitary. This is done by making a snugly fitting plaster cap for each patient. The cap is made to fit low enough on the head to hold the head firmly. Guide marks are placed on the skin and on the cap so that it may be taken off and replaced in the same position.

The plaster cap is prepared by putting one layer of stockinet material over the patient's head. Strips of wet plaster gauze are then molded in conformity with the patient's head. The cap is easily removed even when still slightly wet. Before removal, guide marks are placed on the cap corresponding to the projection of the anterior and posterior sagittal plane, the outer canthus of each eye, and the center of the external auditory meatus.

When the cap is dry, a circular disk is firmly wired to the top of the cap. The plate must be parallel to the plane of the treatment portal centers. The distance of the disk from the top of the plaster cast cap must be known accurately so that the central ray of each roentgen-ray beam will pass through the center of the pituitary fossa. An adjustable metal bolt is attached to the disk on the top of the cap. The bolt is threaded for fixation in the plate of the beam alignment device.

The hole in the disk through which the bolt will be placed has been drilled oversize to allow adjustment. The position of the bolt represents the rotational axis and must be aligned with the pituitary fossa. This is accomplished as follows: With the skull cap on the patient's head, lateral and posteroanterior roentgenograms of the head are taken with a diagnostic roentgen machine. Any deviation between the vertical axis through the sella turcica and the bolt is corrected. The patient is seated upright in a treatment chair and the finished cap is placed on his head. The bolt is inserted in the "rotation" hole mentioned above.

Final adjustments of the beam direction are made as follows: With the patient in treatment position, roentgenograms, using the treatment beam, are taken in at least three directions (right and left lateral, anteroposterior or posteroanterior). Any misalignment in the vertical axis is corrected by further adjusting the position of the bolt. Any misalignment in the horizontal treatment plane is corrected by changing the spacing between the disk of the cap and the plate of the beam alignment framework.

At any subsequent treatment session, the beam is automatically aligned as long as the cap is properly placed on the patient's head and the beam alignment framework is exactly in a horizontal position.

This is checked before each treatment by means of a spirit level. Some movement of the patient may occur after the entire mechanism is fixed in place due to slight flexibility of the whole apparatus. However, since the apparatus is close to the patient's head, the beam literally moves with the patient. This has been ascertained by roentgenograms taken with the treatment beam at different times during one sitting and during different sittings. It has been found that the position of the beam relative to the sella turcica does not vary by more than 1 mm. for any treatment field.

The use of this equipment is not limited to irradiation of lesions in the sella turcica; but can be used for any intracranial tumor as long as the vertical, or rotational, axis and the horizontal, or roentgen-ray beam, axis can be aligned. The size and shape of

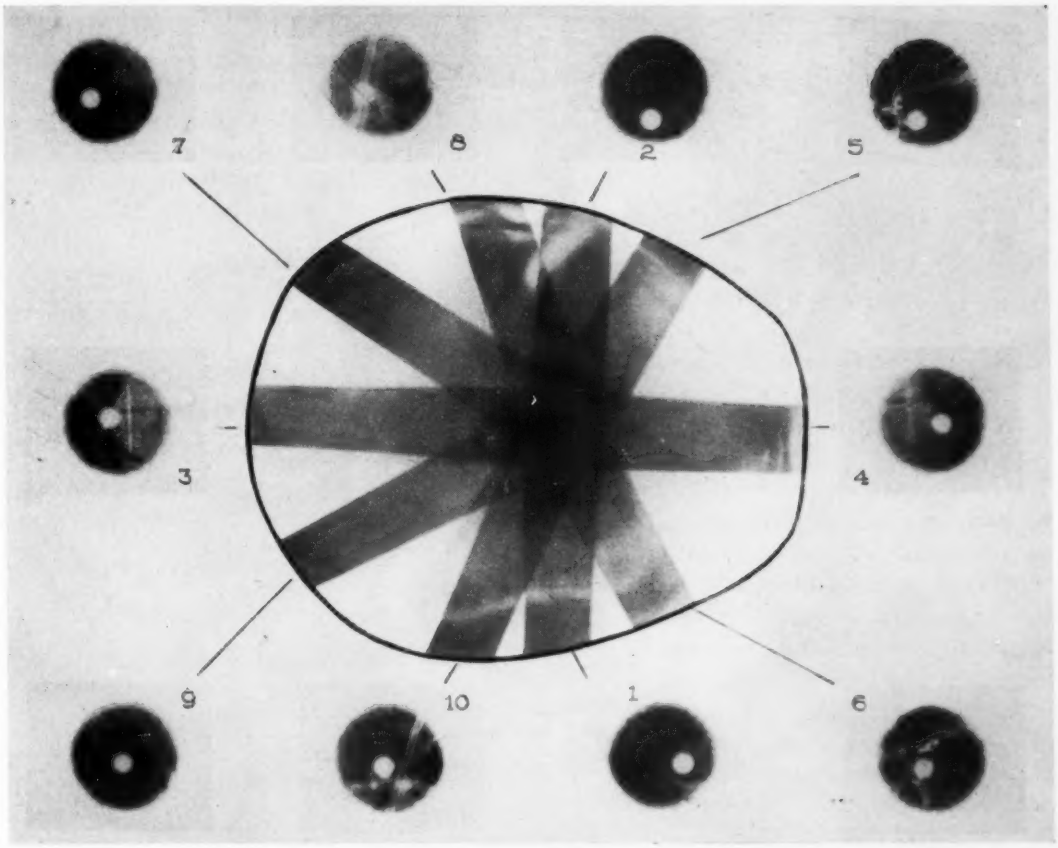

Fig. 3. Pattern formed in the head by the roentgen-ray beams intersecting at the pituitary fossa. The field films of the rice-filled skull are shown with a steel ball placed in the pituitary fossa.

the beam obviously must be adapted to the size of the lesion.

Depth dose measurements to be used for multiple field directed beam therapy differ from those usually found in the literature in that the target-tumor distance rather than the target-skin distance remains fixed throughout the measurements. Consequently, the skin dose to field may vary substantially.

In order to determine the dose distribution in irradiation of the pituitary, the following factors were investigated:

(a) The geometrical pattern in the head formed by the intersecting roentgen-ray beams.
(b) The anatomical landmarks in the path of each roentgen-ray beam.
(c) The depth dose measured in a presdwood phantom and the dose delivered to the sella turcica region in a rice-filled skull and in a wax head phantom.
(d) Depth dose data.
(e) The attenuation of the roentgen-ray beam by the wall thickness of the plaster cap.

(a) The pattern formed by the intersecting roentgen-ray beams was demonstrated by exposing a roentgen film placed between the two halves of the wax head phantom which had been divided at the horizontal plane passing through the center of the pituitary fossa. The exposures were made with the wax head phantom mounted in the beam direction framework so as to simulate treatment conditions. Figure 3 shows this pattern. Figures 4 and 5 show the wax phantom and the rice-filled skull in the beam direction framework.

(b) Field films were taken with the pa-

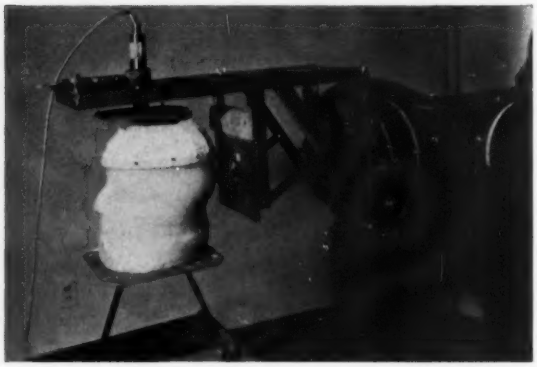

FIG. 4. Wax head phantom in beam direction framework with rate meter probe in position.

tient in each treatment position to localize the bony structures through which each beam passed. Other field films were taken throughout the course of treatment to check accuracy of positioning and thus the validity of the dose estimation. It was found possible to maintain the roentgen-ray beam through the sella with a variation not exceeding 1 or 2 millimeters.

(c) Measurements of the dose to the pituitary region from each field were made in the presdwood phantom, the wax head phantom, and the human skull placed in the beam direction apparatus with the treatment conditions simulated.

The presdwood sheets used were 30 by 30 cm. and 0.635 cm. ($\frac{1}{4}$ inch) thick. Seven centimeters of presdwood backing was used for all measurements and the measuring instrument was a Victoreen rate meter.

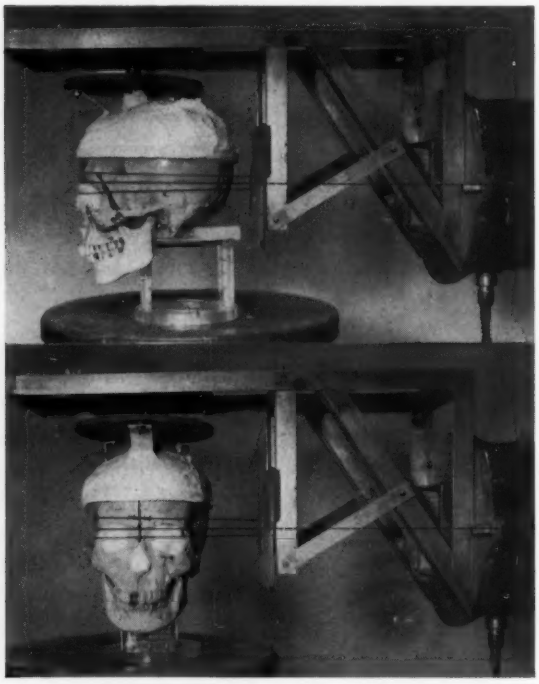

FIG. 5. Shows rice-filled skull in the beam direction framework, shows treatment position and indicates field size.

TABLE I

COMPARISON OF DEPTH DOSE MEASUREMENTS MADE IN RICE-FILLED SKULL, WAX HEAD, AND PRESDWOOD PHANTOMS. 200 KV., 25 MA., HALF-VALUE LAYER 1 MM. CU, FIELD SIZE 3.6 CM. DIAMETER AT 50 CM. OR 4.5 CM. AT 65 CM. (TARGET-TUMOR DISTANCE)

Field	Meter Depth	Presdwood Per cent Depth Dose r/min.*		Rice Skull r/min.*
A. Rice-filled skull (dry rice, orbits filled with wet cotton)				
Anterior	6 cm.	32	16.2	16.0
L. ant. oblique	7	27	13.8	14.5
R. ant. oblique	7	27	13.8	14.3
L. lateral	6	32	16.2	14.8
R. lateral	6	32	16.2	15.6
L. post. oblique	9	20	10.2	10.7
R. post. oblique	10	17	8.7	9.5
Posterior	11	14.5	7.4	8.0
B. Wax head pantom (paraffin)				
Anterior	6.5	29.5	15.3	16.6
L. lateral	6.5	29.5	15.3	17.3
R. lateral	6.5	29.5	15.3	16.5

* Per cent of air r at 50 cm.

A deep water trough was formed in the top of the wax head phantom. The waterproofed rate meter probe could be introduced into this trough to the position of the sella turcica or other positions in the same plane either anteriorly or posteriorly. The rice-filled skull permitted the introduction of the rate meter only into the sella turcica.

Table 1 shows the results obtained from the phantom measurements as well as corresponding values obtained from presdwood measurements for comparison. For convenience, all depth dose values are

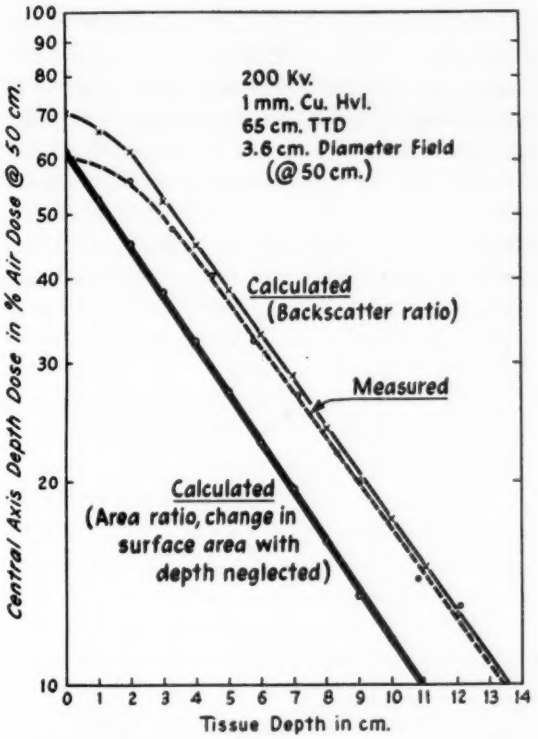

Fig. 6. Calculated and measured central axis depth dose curves for a fixed target-tumor distance of 65 cm. and a field size at the tumor 4.7 cm. in diameter. The depth dose values are expressed as percentages of the air dose at 50 cm.

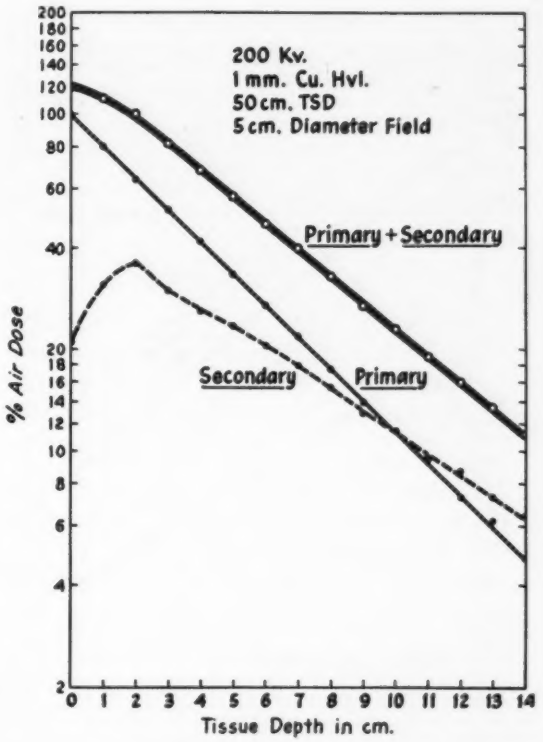

Fig. 7. Central axis depth dose curves using a 5 cm. diameter field showing the division into primary and secondary radiation. (From Loevinger, Wolf and Minowitz.[6])

expressed as percentages of the rate in air at 50 cm. distance, which is the distance at which the roentgen-ray output of the therapy unit used is calibrated daily in this department. The results of the measurements in the presdwood phantom are shown in Figure 6.

(d) Fixed target-tumor distance depth dose curves were calculated from depth dose data available in the literature[6] for the conditions being used. There are six steps to the calculation: (1) The relative intensities of the primary beam and the scattered radiation must be determined for each depth for the smallest field size available (Fig. 7). (2) Then the secondary component must be reduced by a factor which varies with the ratio of the field area desired to the field area available. (3) The third step is to recombine the reduced secondary and the primary beam. (4) Each depth dose value for the new field size must be multiplied by the ratio (T.S.D. +depth)^2/T.S.D.) since the target-tumor distance remained fixed during treatment (Fig. 8). (5) The resulting depth dose values are now plotted against field area, giving a series of points lying on a straight line corresponding to this field area. The points mark the position of each depth (*i.e.*, 1 cm., 2 cm., etc.) on the depth dose axis (Fig. 9).

This process is repeated for slightly larger field areas, and curves connecting the corresponding depth points are drawn. The surface area of the field is used in the above calculations for only one depth, namely the depth such that T.T.D.-depth =T.S.D.*

For any other depth the field area at the surface will be larger because of the greater target-skin distance. For the 3 cm. depth

* The fixed T.S.D. at which the depth dose data were measured.

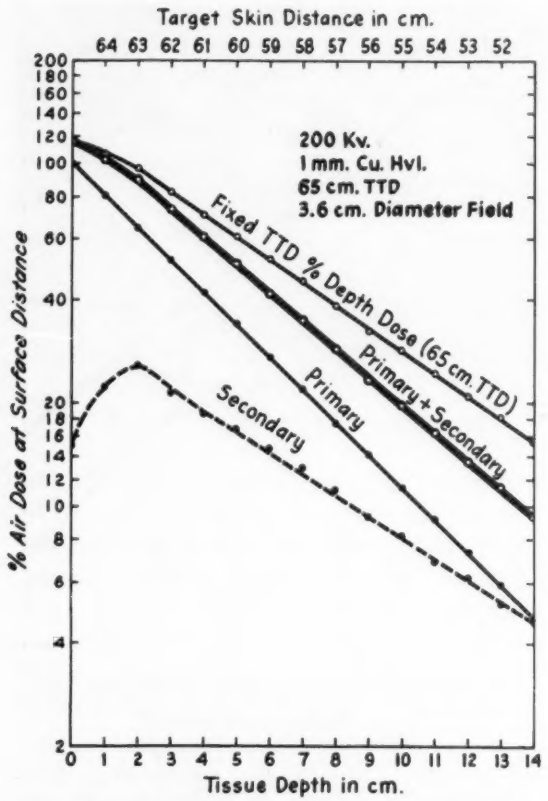
Target Skin Distance in cm.
64 63 62 61 60 59 58 57 56 55 54 53 52
200 Kv.
1 mm. Cu. Hvl.
65 cm. TTD
3.6 cm. Diameter Field
Fixed TTD % Depth Dose (65 cm. TTD)
Primary + Secondary
Primary
Secondary
% Air Dose at Surface Distance
Tissue Depth in cm.

FIG. 8. The secondary of Figure 7 has been reduced by the backscatter ratio factor to the secondary of a 3.6 cm. diameter field and added to the primary of Figure 7 to give the calculated central axis depth dose curve for a 3.6 diameter field size. Finally, the inverse square factors have been eliminated to give a fixed target-tumor distance central axis depth dose curve.

dose, for example, the 3 cm. depth dose from a field of an area $(\text{T.T.D.}-3)^2/\text{T.S.D.}^*)$ times the surface area must be used. When the proper depth doses are found by this method, they may be connected and will then form a smooth curve as shown in Figure 9.

For comparison with the measured depth dose values, the calculated depth dose values are expressed as percentages of the 50 cm. air dose. A comparison between the calculated and measured depth dose curve may be seen in Figure 6 for a field size of 3.6 cm. diameter at 50 cm. T.S.D. Similar calculations must be made to obtain the surface and exit dose values for this type of therapy.

Determination of the Area Scatter Relationship. To a first approximation, the relationship between field area and secondary or scattered radiation produced at the center of the field is the same as the field area-backscatter relationship which is usually available. Thus, if it is desired to calculate the secondary contribution from a 3 cm.^{2} field using the data from a 5 cm.^{2} field, the 5 cm.^{2} secondary values for each depth are multiplied by the ratio:

$$\frac{3 \text{ cm.}^2 \text{ backscatter}}{5 \text{ cm.}^5 \text{ backscatter}}.$$

It should be noted that between 3 cm.^{2} and 30 cm.^{2} field areas, the backscatter ratio changes from 1.5 to 0.7 per cent/cm.^{2} for 200 kv., 1 mm. Cu half-value layer. Thus, it is not feasible simply to use the ratio of areas of the two fields as a multiplying factor. The result of doing this, and

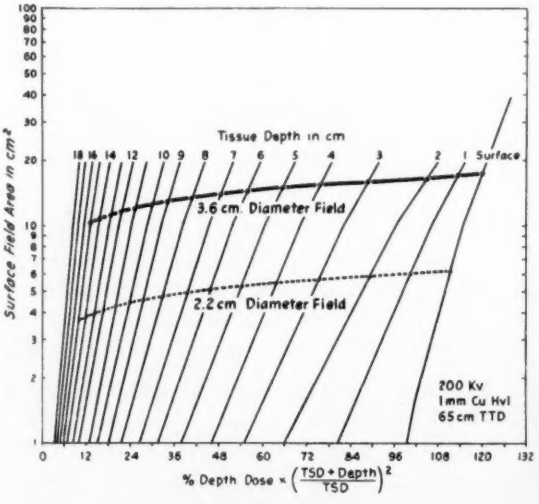
Surface Field Area in cm²
Tissue Depth in cm
18 16 14 12 10 9 8 7 6 5 4 3 2 1 Surface
3.6 cm. Diameter Field
2.2 cm. Diameter Field
200 Kv
1 mm Cu Hvl
65 cm TTD
% Depth Dose × ((TSD + Depth)/TSD)²

FIG. 9. Fixed target-tumor distance depth dose curves plotted against surface field area. The vertical lines give the (1 cm., 2 cm., etc.) depth dose as a function of the surface field area. The last mentioned curve in Figure 8 appears here as a series of points along the horizontal line representing 10.2 cm.^{2} surface field area. For a depth of 15 cm. this area is correct, since the target-skin distance equals 65–15 equals 50 cm. For smaller depths the surface field area is increased due to the increased target-skin distance and therefore each depth dose must be found for a different field area. This is shown for a 2.2 cm. and 3.6 cm. diameter field (at 50 cm.)

also of ignoring the changing surface area, may be seen in Figure 6.

For the greatest accuracy, the area-backscatter measurements should be made using circular fields. Figure 10 shows a typical area-backscatter curve. This has been plotted on logarithmic graph paper to cover the range of area and also to flatten out the very steep initial slope of the curve. Also in Figure 10 is a curve showing the backscatter/area ratio in per cent/cm.^{2} for various field areas. The initial rise probably represents the building up of electronic equilibrium in the thimble chamber which is more than half the size of the field at these small areas.

(e) The average plaster cap thickness was 3/16 inch. Measurements showed that the beam was diminished by approximately 10 per cent in passing through this thickness of dry plaster. Where practicable, the plaster was not placed in the beam, or, if so, was made as thin as possible at that place.

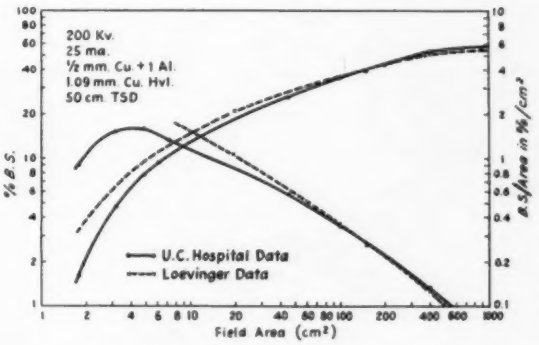

Fig. 10. Backscatter-field area curve plotted on logarithmic paper to spread out the low area part of the curve. Also curve showing the backscatter area ratio in per cent/cm.^{2} as a function of field area. The initial rise probably represents the building up of electronic equilibrium in the thimble ionization chamber, which is more than half the size of the field at these small areas.

REPORT OF CASES

Five patients were treated by the multiple small field, directed beam technique in 1949 and 1950. One patient had a pituitary adenoma, 1 had a glioma at the chiasma of the optic nerve. Three patients had disseminated malignancies: 1 from melanoma, the other 2 from carcinoma of the breast.

The objectives of reduction or interruption of pituitary function in disseminated carcinoma was the stimulus for this entire study. At the same time, the study fitted into our more comprehensive program of directed beam therapy.

The following case histories are presented to demonstrate the beam direction set-up; to show the physical factors of treatment, the radiation dose on each skin field, and the calculated aggregate tissue dose. No attempt is made here to evaluate the results of treatment.

Fig. 11. Shows treatment set-up on Case 1 (patient N. T.).

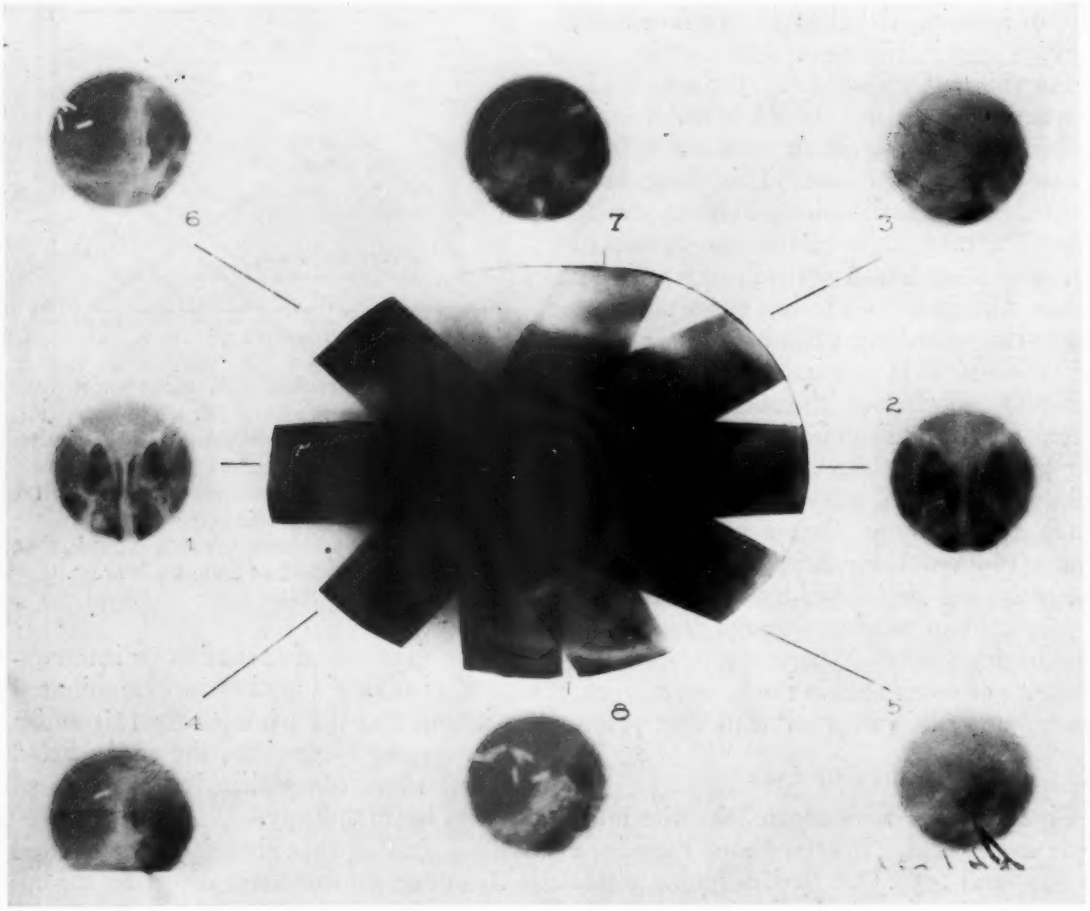

FIG. 12. Shows irradiation field pattern and field films on Case I (patient N. T.).

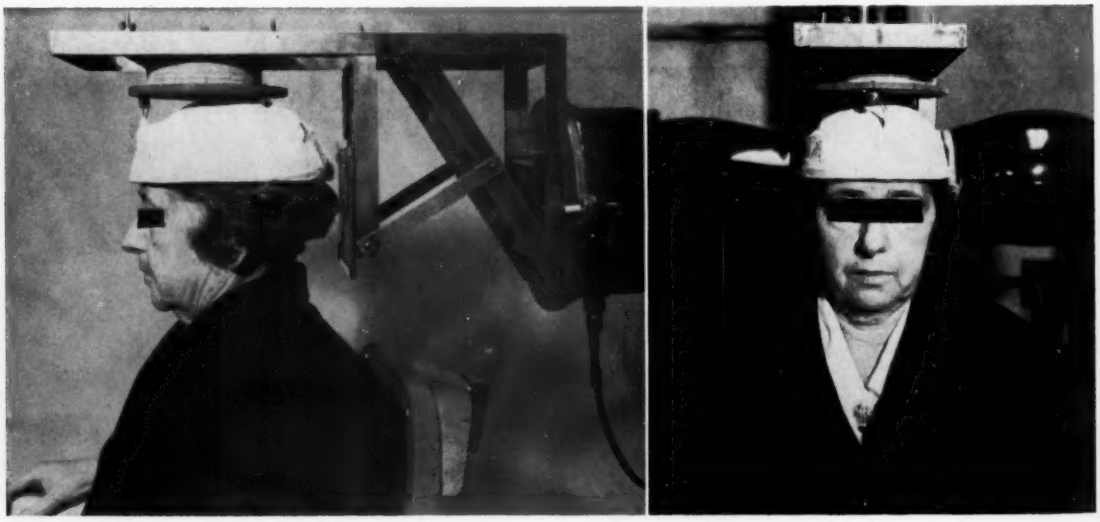

FIG. 13. Shows treatment set-up on Case III (patient J. C.).

Case I. N. T., a white male, aged six, was seen in February, 1949, with complaints of poor vision getting progressively worse for the past six months. Surgical exploration and biopsy confirmed the diagnosis of a glioma of the optic nerve, located in the suprasellar region. The tumor could not be completely removed surgically. In April, 1949, the patient received a course of roentgen therapy. Physical factors were: 200 kv., 25 ma., half-value layer 1.08 mm. Cu, target-skin distance 78 cm. Treatment was given to two temporal fields, 7 by 7 cm., one on each side and centered at the suprasellar region. Thirty-one treatments were given in thirty-five days. A calculated tissue dose of 3,580 r at the depth of 7.5 cm. was delivered.

In September, 1949, the patient complained of increasing thirst and polyuria and noted a little difficulty in walking. A craniotomy performed at that time revealed a 2 cm. mass arising from the right optic nerve.

All of the tumor could not be removed, and it was decided to give a second course of radiation therapy using the multiple beam technique. Therapy was directed to the region surrounding the optic chiasma.

Physical factors were 200 kv., 25 ma., half-value layer 1.08 mm. Cu. Twenty treatments were given in thirty days. Figure 11 shows the treatment set-up on this patient. Figure 12 shows the irradiation pattern and field films of this patient. The patient was treated through eight fields, sparing the skin in previously treated areas. The dose delivered to each field and the aggregate calculated tumor dose were as follows:

Fields	*Air r at 50 cm.*	*Skin r at actual distance*	
1	1,900	1,615	
2	1,600	1,360	
3	1,600	1,120	
4	1,600	1,408	3,479 r calculated
5	1,600	1,408	tumor dose
6	1,600	1,280	
7	1,600	1,280	
8	1,600	1,615	

Skin roentgens are lower than air roentgens because the target-skin distance is 53–56 cm., whereas air dose is measured at 48 cm.

The treatment course was uneventful, and since therapy the patient has remained in good condition. Epilation occurred in the treated areas; but after three months, hair growth was entirely restored. He was last seen in June, 1951, in excellent general health. His progress in school is average.

Case II. L.D., a white female, aged thirty-seven, was first seen in August, 1950, complaining of severe frontal headaches and distorted vision gradually getting worse during the last two years. These symptoms were associated with the coarsening of the facial features and some enlargement of the extremities, particularly the hands. The patient had not menstruated for three years. Basal metabolic rate in August, 1950 was minus 21 per cent. Roentgenograms of the skull showed a concentric enlargement of the sella to about two times normal size. The diagnosis of acromegaly was made and radiation therapy was decided upon.

Directed beam, multiple field technique was used with the physical factors: 200 kv., 25 ma., half-value layer 1.08 mm. Cu; thirty-two treatments were given in forty days. Treatments were given through seven fields of 3 cm. diameter. The dose to each field and the aggregate calculated tumor dose were as follows:

Fields	*Air r at 50 cm.*	*Skin r at actual distance*	
1	2,360	2,000	
2	2,300	1,850	
3	2,500	2,030	5,050 r calculated at
4	2,700	2,400	the pituitary
5	2,770	2,200	
6	2,030	1,650	
7	2,760	2,450	

The treatment course and post-treatment period were uneventful. After temporary epilation, hair growth has been completely restored.

Case III. J. C., a white female, aged fifty-nine, with disseminated bilateral adenocarcinoma of the breast. She first noticed a painless lump in her left breast four years previously. A radical mastectomy was performed, and roentgen therapy was given later for local recurrences.

When first seen by us, the patient had widespread carcinosis. Irradiation directed to the pituitary fossa was started on November 17, 1949. Physical factors were: 200 kv., 25 ma., target-skin distance 52 to 56 cm., half-value layer 1.08 mm. Cu. Field size was 2 cm. in diameter. Treatment was given through ten

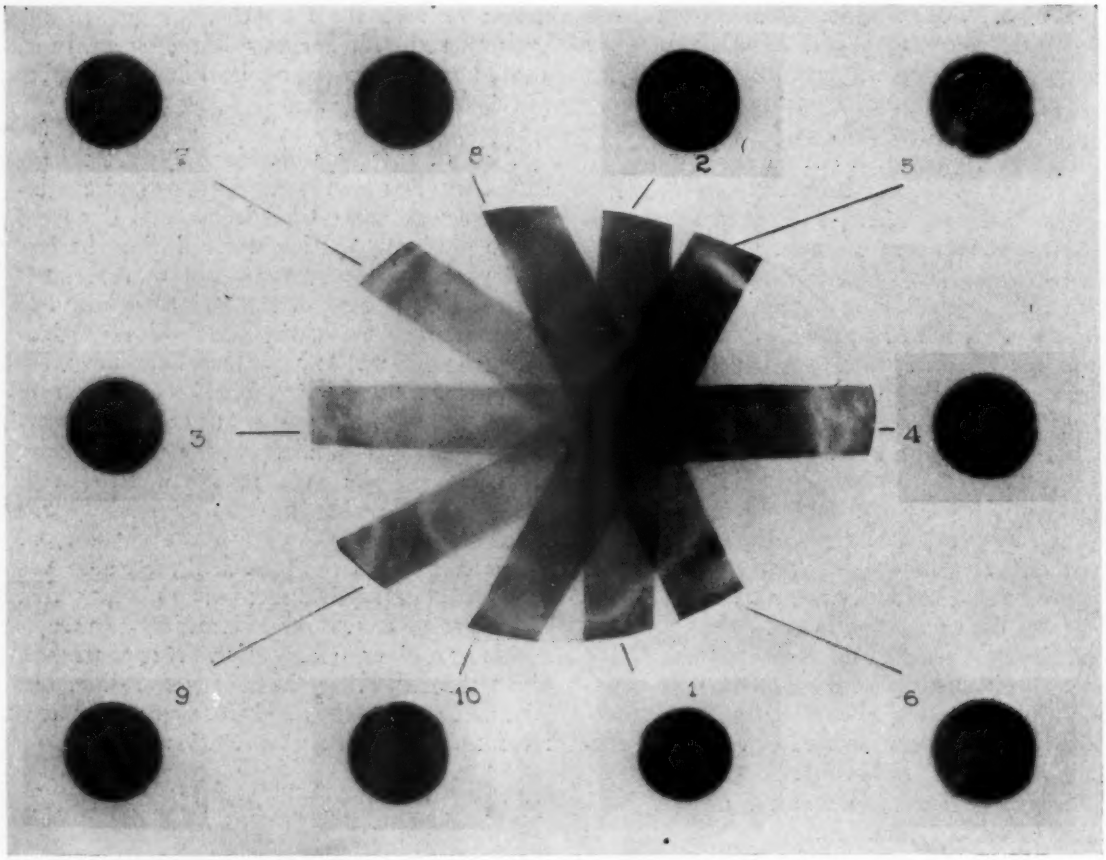

FIG. 14. Shows irradiation field pattern and field films on Case III (patient J. C.).

fields, forty-seven treatments in sixty-two days. The dose to each field and the aggregate calculated tissue dose were as follows:

Field	*Air r at 50 cm.*	*Skin r at actual distance*	
1	5,200	4,360	
2	5,200	4,360	
3	5,200	4,790	
4	5,200	4,200	10,000 r tissue dose
5	5,200	4,290	calculated at
6	5,200	4,290	the pituitary
7	4,800	4,270	
8	4,800	4,580	
8	4,800	4,270	
10	4,800	4,580	

Figure 13 shows the treatment set-up and Figure 14 shows the irradiation pattern and field films on this patient.

Treatment was well tolerated. Epilation developed in the areas of irradiation, with moderate erythema of the skin. The erythema subsided about fifty days after treatment. The treatment apparently produced no effect on the disease, and the patient died four and a half months after irradiation of the pituitary.

Autopsy performed three hours after death showed widespread carcinosis. Microscopic examination of the pituitary showed the anterior lobe to be cellular with a normal ratio of cells. Except for a slight degree of interstitial fibrosis, nothing was observed in the pituitary which is not seen in persons of the same age as this patient.

CASE IV. G. M. H., a white female, aged thirty-eight, with disseminated melanoma arising from a plantar pigmented mole. She had had a pigmented mole on the plantar surface of the fourth left toe since birth. Six years previously, in the fourth month of her second pregnancy, there was a change in the appearance of the lesion and the toe was amputated. The diagnosis on microscopic examination was malignant melanoma. Subsequently, new melanotic

nodules appeared on the leg. These were locally excised.

Physical examination on admission revealed a well nourished, well developed woman. The left leg was swollen with edema; and there were numerous melanotic nodules, some measuring up to 2 cm. in diameter, over the whole extremity. The left inguinal region was filled with firm, nontender masses extending above the inguinal ligament. It was decided to attempt to ablate the pituitary by irradiation. Irradiation directed to the pituitary fossa was started in September, 1949.

Physical factors were: 200 kv., 25 ma., half-value layer 1.08 mm. Cu, target-skin distance 53 to 55 cm. Field size was 2.2 cm. Treatment was given through ten fields, centering and crossing at the pituitary fossa. Fifty treatments were given in sixty-three days, three fields being treated each day. The dose to each field and the aggregate calculated tissue dose were as follows:

Fields	*Air r at 50 cm.*	*Skin r at actual distance*	
1	4,400	3,790	
2	4,400	3,790	
3	4,400	4,110	
4	4,400	3,610	
5	4,400	3,670	8,510 r calculated at the pituitary
6	4,400	3,670	
7	4,400	4,000	
8	4,400	3,310	
9	4,400	3,270	
10	4,400	3,310	

Figure 15 shows the treatment set-up, and Figure 16 shows the irradiation pattern and field films for this patient. Treatment was well tolerated. Epilation and moderate erythema developed in the irradiated areas. Erythema subsided about the sixtieth day following treatment. The melanotic lesions of the left leg increased steadily in size, and many became ulcerated. There was progressive weakness and loss of weight. Ascites appeared approximately two months following the completion of irradiation, and melanin was observed in the urine. The patient died three months following completion of irradiation of the pituitary gland.

Microscopic examination of the pituitary gland showed a delicate, well formed anterior and posterior lobe without cellular or vascular changes. The ratio of cells within the anterior lobe appeared to be within normal limits. Examination of all the endocrine glands showed no abnormalities except where tumor was invading. Both ovaries were replaced by tumor. Examination of the base of the brain, internal carotids, pineal body, cranial nerve ganglia, optic chiasm, and floor of the third ventricle revealed no abnormalities.

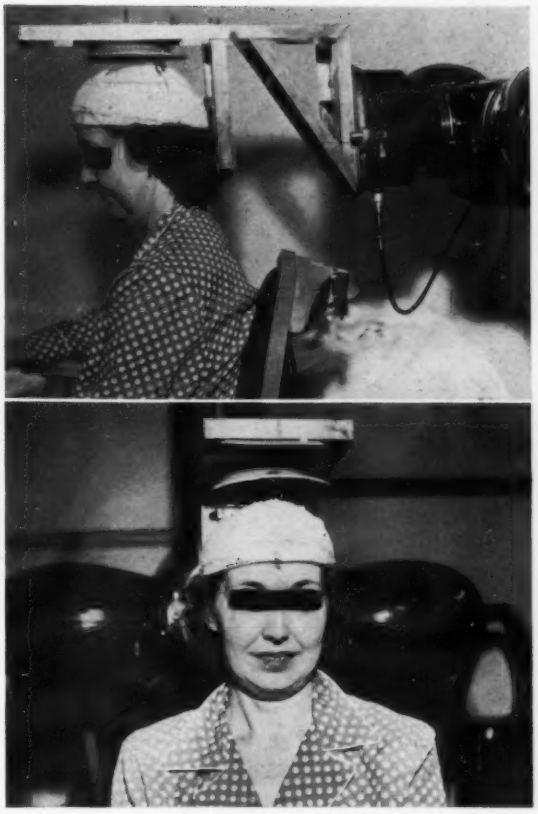

Fig. 15. Shows treatment set-up on Case iv (patient G. M. H.).

Case v. M. K., a white female, aged sixty, with disseminated adenocarcinoma of the right breast. At the age of fifty-five she had noted a painless nodule in the right breast. Radical mastectomy was performed two months later, and she remained clinically free of disease until two months before admission. Recurrence was first noted in the right supraclavicular region, followed by the appearance of hard, nontender subcutaneous nodules along and near the surgical scar.

Roentgen examination failed to reveal bone or pulmonary metastases. Biopsy of one of the subcutaneous nodules revealed metastatic adenocarcinoma compatible with the histopathological appearance of the primary carcinoma

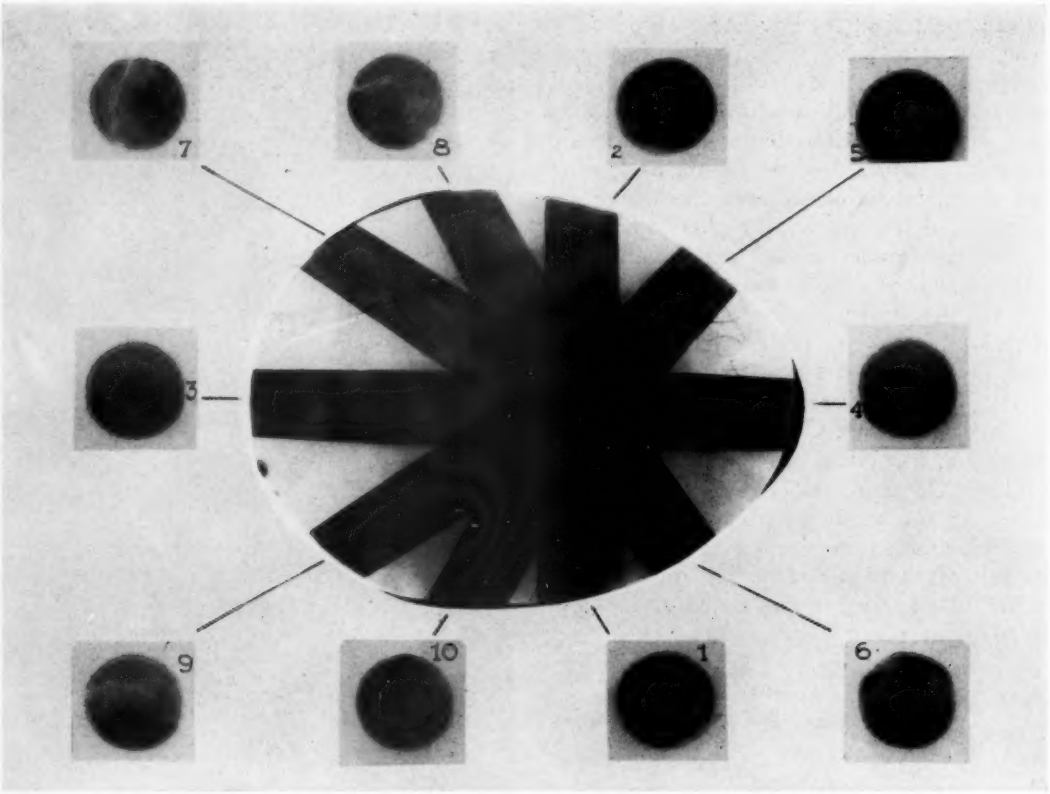

FIG. 16. Shows irradiation field pattern and field films on Case IV (patient G. M. H.).

FIG. 17. Shows treatment set-up on Case V (patient M. K.).

of the breast. It was decided to irradiate the pituitary, commencing on April 22, 1949. Physical factors were: 200 kv., 25 ma., half-value layer 1.2 mm. Cu, target-skin distance 53 to 56 cm. Field size was 2.5 cm. in diameter. Treatment was given through ten fields centering and crossing at the pituitary fossa. Forty-seven treatments were given in sixty-two days. The dose for each field and the aggregate calculated tissue dose were as follows:

Fields	*Air r at 50 cm.*	*Skin r at actual distance*	
1	4,320	3,680	
2	4,040	3,470	
3	4,290	3,800	
4	3,180	2,900	
5	4,400	4,090	8,150 r calculated at the pituitary
6	4,200	4,010	
7	4,000	3,710	
8	3,180	2,900	
9	3,890	3,440	
10	3,640	3,060	

Figure 17 shows the treatment set-up, and

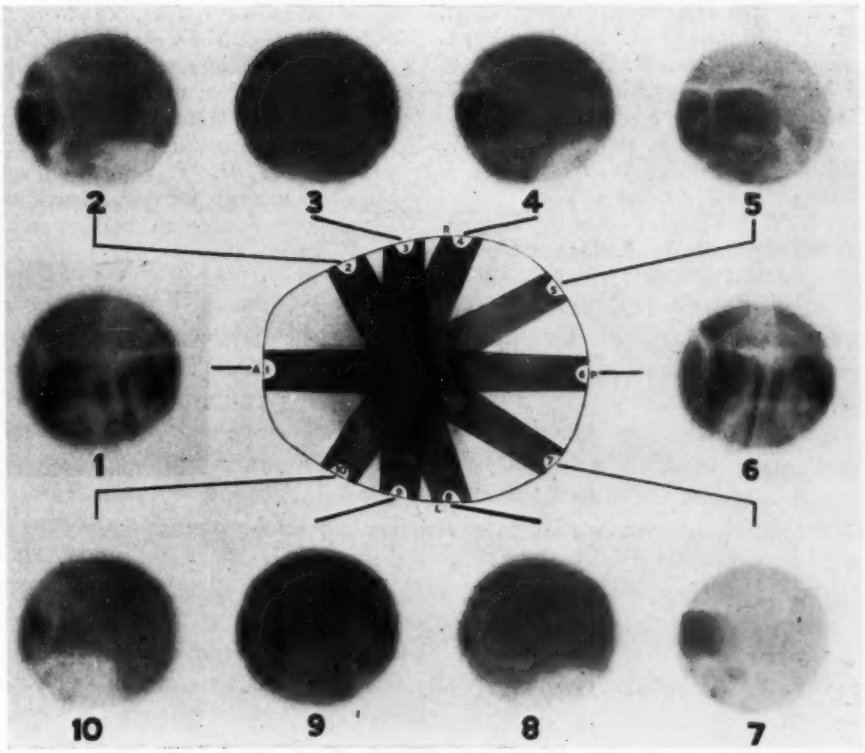

Fig. 18. Shows irradiation field pattern and field films on Case v (patient M. K.).

Figure 18 shows the irradiation pattern and field films for this patient. The patient tolerated the procedure without complications.

Epilation, erythema, and tanning of the skin subsequently developed over irradiated areas. Measurements and clinical observations of the metastatic lesions did not reveal any effects on the tumor.

The patient was last seen in November, 1951. Persistent epilation in the fields of irradiation was observed. The skin in these areas appeared slightly depigmented, but no atrophy and no telangiectatic vessels were seen. In the intervening period, the patient received local roentgen treatment to individual metastatic skin nodules at the Mallinckrodt Institute of Radiology in St. Louis.

SUMMARY

1. The principles of small field, directed beam therapy with roentgen rays are discussed.

2. A beam direction device is described and its application is illustrated.

3. Dose measurements and calculations are explained.

4. Five case histories are presented to illustrate the technique.

It is our conclusion that the technique herein described affords a means of accurate focal irradiation of pituitary tumors and other intracranial lesions.

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THE TREATMENT OF MODERATELY ADVANCED CARCINOMA OF THE PROSTATE WITH RADIOACTIVE GOLD*

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CARCINOMA of the prostate is a common malignant disease with a poor prognosis.[3,7] Its incidence is increasing due to the ageing of the male population and the fact that it is being recognized more frequently by the profession as a whole, but the cure rate has not been appreciably raised in the past few decades. This is in contrast with the advances in the successful attack on malignant tumors elsewhere in the body, notably the central nervous system, lung and gastrointestinal tract. This is because it is generally a "silent" tumor and produces symptoms of urinary obstruction or pain relatively late in the course of the disease. Moore discovered carcinoma of the prostate in 21 per cent of autopsies on men over the age of forty years. The number of men in the United States living beyond sixty-five years doubled between 1911 and 1935, and the male life expectancy at birth increased from 48.3 to 63.4 years from 1911 to 1941. This ageing factor has increased the incidence of the disease until it is now estimated that at least 3 million men in this country have carcinoma of the prostate. From a glance at these figures, one gathers some idea of the problem that confronts us.

Our anatomic classification of carcinoma of the prostate according to extent of disease is as follows: (1) the tumor is small, freely movable and confined within the capsule of the gland; (2) there is extension beyond the capsule but no evidence of metastasis outside the pelvis; (3) evidence of distant metastasis. Because of the silent nature of the disease, only a small percentage falls into the first classification—in our hospital about 5 per cent. The second category includes 40 to 60 per cent, while the remainder show distant metastasis and are put in the third group. Young found 3.4 per cent curatively operable while Barringer considered 4.5 per cent localized in the gland.

We are at present in agreement with those who state that the best treatment for cases in Group 1 is radical surgery, but one of the difficulties is an accurate preoperative determination of the extent of the lesion. For example, Jewett found that of 89 cases without clinical evidence of spread, 32 had microscopic extension beyond the prostate. Our own experience is similar and we have found ourselves in error in both directions, *i.e.*, either greater or less involvement than was determined by digital examination by as much as 200 per cent. Assuming, then, an operability rate of 5 to 10 per cent, we are still confronted with 40 to 60 per cent of the cases who have no demonstrable metastases and yet who are obviously beyond hope of "cure" by surgical means.

Since the epoch-making discovery of Huggins of the effects of female hormones or orchiectomy on carcinoma of the prostate, most urologists and radiologists have been content to abandon hope of curing these patients and have treated them palliatively and expectantly. This attitude is justified when metastases to bone are evident on roentgenographic examination, or are suspected from phosphatase determinations or where there is a huge mass with evident spread outside the gland and throughout the pelvis.

Between this group and the obviously operable cases, however, there is a large number of cases who, in our opinion, should be given the benefit of some attempt at

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cure. If surgery for these patients is insufficient, perhaps some combination of surgery and irradiation would be worth while.

Acting on this thought over the past few years, we have tried roentgen therapy alone, or roentgen treatment and radon seeds with only an occasional good result. Many others have used radiation in its various aspects.[1,2,4,8,9] None considered that radiation as then used was the answer to this serious problem. Sufficient radiation can be given to destroy the tumor, but adjacent normal structures, notably the rectum and bladder, suffer from too much radiation and are grossly injured. With the advent of Au^{198} whose main radiation component is beta rays, the prostate seemed an admirable site in which to try this new isotope.

Our first patient was a man, aged seventy-two, who had had a proved carcinoma of the prostate for five months. He had had a transurethral resection for obstruction and had been on stilbestrol following proof of the lesion. His tumor was to have been implanted with radon and supplemented with external irradiation, but the calculated amount of radon was deemed too dangerous and too expensive. Since we had some radioactive gold on hand, this was used, and in three weeks the large, hard mass was impalpable. Ninety-two days after the injection, biopsies were negative for carcinoma. This case led us to believe that the inoperable carcinoma of the prostate without metastasis might conceivably be just the group for this type of therapy.

Since the clinical material of this kind which we see is fairly abundant, we have been able to treat 129 selected cases in the past seventeen months. We have tried to select those cases which fell into Group 2, but 2 cases were later discovered to have had metastasis at the time the gold was injected and 3 cases have developed metastases since injection.

It is far too early, of course, to show anything except a trend with this type of treatment, but immediate results have been so promising that we are constrained to continue the experiment, since no other method offers anything beyond palliation.

METHOD

A standard retropubic approach is made to the prostate. Palpation of the structure through the unopened bladder and of the pelvis retroperitoneally gives us an idea of the extent of the disease and the size of the lesion. We have been amazed at the discrepancy between our preoperative estimate of the size and extent of the disease and conditions as found at time of operation. Preoperative estimates are frequently in error as much as 100 per cent and have been off as much as 300 per cent in rare instances. Bleeding is controlled and the bladder is opened. A suprapubic catheter is placed in the bladder and carried to the exterior through a stab wound above the original incision. All possible operative procedures are carried out before the actual injection of the gold is begun. This prevents unnecessary exposure and allows the operating team to complete the procedure and leave the vicinity of the patient as rapidly as possible. This is undoubtedly one of the most important factors in the protection of personnel from excessive radiation. After estimation of the amount of tissue involved by malignancy, the radiologist and urologist collaborate on the plan of procedure. The total dosage to be used and the number and location of the various instillations are all decided before any actual injection is begun. It is then the duty of the radiologist to measure the amount of radioactivity desired and dilute it to the proper total volume. He then loads the protected syringe and passes it to the urologist who does the actual injecting. Usually from 8 to 16 cc. of the diluted material are injected into the prostate from within the bladder. This allows one to reach the apex of the prostate and inject quite thoroughly around the urethra as well as the two lateral lobes. After this the periprostatic region and the seminal vesicles

are injected transvesically and extravesically. Any palpable nodes are also injected at this time. In 2 instances we have found large masses extending from the prostate to the pelvic wall—a condition similar to a frozen pelvis in carcinoma of the cervix. In both of these cases the extensive malignant mass was infiltrated as thoroughly as possible, with rather surprising clinical results. Following the injection, the bladder and incision are closed as rapidly as is consistent with good surgical technique. A retention catheter is passed through the urethra in order to facilitate drainage.

DOSE

Initially we set up a somewhat arbitrary dosage scale. If the estimated amount of involved tissue was between 20 and 35 grams, we used 3 millicuries per gram; if 35 to 70 grams, 2 mc. per gram; if 75 to 150 grams, 1 mc. per gram of tissue. For a mass of over 150 grams we used less than 1 mc. per gram. Our total dose then varied from about 20 mc. to 150 mc. Because of certain complications, which will be discussed later, we thought it advisable to reduce this dose. The last 25 or 30 cases have been done with a millicurie per gram ratio of 1 to 1, or 1.6 to 1. The cases treated with this dosage who have returned have not been completely satisfactory, and the regression of their tumors has not been so complete as with our former dose scale. It is our impression at the present time that a moderate sized prostate, between 30 and 60 grams, is probably best treated with a millicurie per gram ratio of 2 to 1. The dose employed in the injection of the seminal vesicles has also been reduced. Originally, we used as high as 20 mc. in each seminal vesicle, but this seemed to increase the number and severity of the rectal complications which we encountered. That dose now is limited to between 5 and 10 mc., depending upon the extent of the disease. We have been using as a diluent, a mixture of 100 cc. of normal saline, 100 turbidity units of hyaluronidase, and 1 cc. of 1 to 1,000 epinephrine. From animal experimentation and from the 2 autopsies which we have obtained on patients dying in the first week following injection, it seems that this gives somewhat better distribution of the injected material than does saline solution alone. We think that it also increases the amount of gold which is absorbed into the circulation, as is evidenced by higher counts over the area of the liver and spleen. However, we have had no instance of liver damage that could be detected either clinically, by liver function tests or pathologic examination.

PROTECTION

The principal activity of Au^{198} is beta radiation of 0.98 mev., but there are also two gamma rays of 0.12 and 0.41 mev. respectively. The material has a half-life of 2.7 days. The problem of protection is therefore aimed at these two components. The main protective factors stressed in these experiments are (1) shortness of exposure time and (2) adequate lead barrier where possible. Thus, the material is completely surrounded with adequate lead until the prostate is exposed and an estimate of the advisable dose is made. The material is then withdrawn with a syringe protected with 5 mm. of lead. Only the eyes and the head above the eyes of the radiologist are exposed during this procedure. Mixture with the NaCl solution, the hyaluronidase and epinephrine is made in a lead-protected medicine glass, and from this it is withdrawn into a 5 cc. Luer syringe which is protected by 15 mm. Pb. The needle is connected to the syringe by a lead tube. This allows some flexibility and at the same time gives a spacer and some protection. Careful monitoring of the radiologist, surgeons, nurses, patient, sponges and drapes has been done repeatedly. The radiologist, who is in a position to receive more radiation than any other member of the team, has always received less than 150 milliroentgens per week. For the surgical team, speed is important. Au^{198} can be washed from instruments and gloves very

TABLE 1

SUMMARY OF FIRST FIFTY CASES TREATED WITH Au^{198}

Case No.	Estimated Weight of Prostate in Grams	Millicuries Au^{198} Used	Mc. per Gm.	Days Till Prostate Impalpable	Complications	Clinical Report		
						Alive		Dead
						Physical	Biopsy	
1	35	70	2.0	92	None	18 mo.; neg.	18 mo.; neg.	
2	21	64	3.0	58	None	12 mo.; neg.	6 wk.; pos.	
3	60	75	1.25	31	None	16 mo.; neg.	16 mo.; neg.	
4	20	70	3.5	14	Rectal ulcer at 8 mo.; colostomy necessary	15 mo.; neg.	7 mo.; neg.	
5	20	70	3.5	7	Rectal ulcer at 6 weeks. Healed with stricture in 6 mo.	13 mo.; neg.	None	
6	150	120	0.8	32	None	12 mo.; neg.	9 wk.; pos.	
7	90	100	1.1	11	None			11 months. Died with disease
8	75	100	1.3	15	None	11 mo.; neg.	2 wk.; pos.	
9	140	140	1.0	Remained palpable and regrowing 63 days	None			3 mo. Died embryonal cancer of the testis with metastasis
10	250	160	0.66	Still hard and fixed 75 days	Mild diarrhea at 1 wk. lasted 2 wk.			4 mo. Died with disease
11	45	125	2.8	21	Diarrhea and blood for 20 days			6 wk. with cancer of esophagus—18 mo. duration
12	50	94	1.9	60	Rectal stricture 3 mo. controlled with dilatation	12 mo.; neg.	11 mo.; neg.	
13	100	67	0.67	15	Rectal irritation 11 days. Stricture at 4 mo. Dilated	10 mo.; neg.	4 mo.; pos.	
14	80	67	0.84	Nodule persisted	None	9 mo. nodule; 13 mo. no symptoms	9 mo.; neg.	
15	70	140	2.0	28	Rectal irritation for 3 weeks			6 weeks—at home. Cause not determined
16	50	106	2.1	12	None	11 mo.; neg.	3 mo.; neg.	
17	150	106	0.71	26	None			6 weeks. Pelvic abscess. Viable tumor
18	75	100	1.3	15	Rectal ulcer and fistula 5 mo. Colostomy with relief	6 mo.; neg.	7 wk.; pos.	
19	140	140	1.0	21	Rectal irritation and fibrosis 2 mo.			4 mo.—at home. Probably tumor
20	250	160	0.66	29		12 mo.; neg.	3 wk.; pos.	
21	580	174	0.3	Pelvis full. Never became impalpable	None			Date and cause unknown
22	40	95	2.7	7	Irritation with blood for 10 days			3 mo. sudden death. Probably cerebrovascular accident
23	30	90	3.0	21	None	9 mo.; neg.	3 wk.; neg.	
24	90	150	1.66	Nodule left reinjected at 8 mo.	Irritation with blood for 10 days	11 mo.; neg.	8 mo.; pos.	
25	85	124	1.5	14	Irritation			Date and cause unknown
26	80	120	1.5	28	None	8 mo.; neg.	3 wk.; pos.	
27	55	88	1.6	42	Rectal irritation for 3 weeks			5 mo. Cause unknown
28	35	105	3.0	14	Slight irritation for 5 days	6 mo.; neg.	3 wk.; neg.	
29	65	130	2.1		None			7 days; pulmonary embolism
30	90	118	1.3	14	None	6 mo.; neg.	None	
31	50	100	2.0	42	None	9 mo.; neg.	None	
32	35	97	2.8	120	None	10 mo.; neg.	None	
33	50	95	1.9	Nodule left	None	1 mo.; nodule	None	

TABLE 1—*Continued*

Case No.	Estimated Weight of Prostate in Grams	Millicuries Au^{198} Used	Mc. per Gm.	Days Till Prostate Impalpable	Complications	Clinical Report		
						Alive		Dead
						Physical	Biopsy	
34	80	147	1.8	Residual	Slight rectal irritation. Bladder calculi. Slough	9 mo.; neg.	9 mo.; pos.	
35	70	99	1.4	120	Irritation for 5 days	7 mo.; neg.	3 wk.; pos.	
36	55	99	1.8	21	Ulceration and stricture 4 mo. Still present 9 mo.	9 mo.; neg.	3 wk.; pos.	
37	35	80	2.3	7	None	9 mo.; neg.	None	
38	45	108	2.4	28	Ulceration, 1 mo. Severe pain. Colostomy			4 mo.—ruptured peptic ulcer. Postmortem—neoplasm
39	60	115	1.9	14	Drug reaction, 3 wk. Agranulocytosis			4 wk. Drug reaction
40	20	60	3.0	21 days impalpable; 8 mo. regrowing	Perineal injection on 4 occasions since			9 mo. Sudden death but had neoplasm
41	30	70	2.6	21	None	5 mo.; neg.	3 wk.; pos.	
42	45	90	2.0		Incomplete follow-up			
43	145	140	0.93	Nodule—8 mo. 2 perineal injections	None	8 mo.; nodule	8 mo.; pos.	
44	30	90	3.0	120	None	5 mo.; neg.	3 wk.; neg.	
45	20	45	2.25	14	None	1 mo.; neg.	3 wk.; neg.	
46	40	75	1.9	90	None	8 mo.; 0.5 cm. nodule	None	
47	40	80	2.0	Nodule—6 mo. Perineal injection	None	7 mo.; metastases. Doing poorly	None	
48	30	60	2.0	60	None	7 mo.; neg.	None	
49	10	25	2.5	Nodule—4 mo. Perineal injection	None	4 mo.; still cancer	None	
50	140	100	0.7	Some regression. Growing again 4 mo. Perineal injection	None	6 mo.; neg.	4 wk.; pos.	

easily, but with great difficulty from the skin. Therefore, rubber gloves should always be worn when handling the material. Contaminated dressings or drapes and small amounts of unused radioactive material are taken to the radiation laboratory for storage until it is safe to dispose of them to the laundry or down the drain. The first urine is also monitored for safety before being discarded.

RESULTS AND COMPLICATIONS

Thus far, 50 cases of carcinoma of the prostate have been treated with radioactive gold and have been followed for from six to seventeen months. The average age of these patients was 68.9 years, with a median age of 69.2. The youngest was forty-six and the oldest was eighty-six. Five of these patients had had one or more transurethral resections for relief of obstruction prior to the treatment with Au^{198}. Twenty-four had had hormonal therapy in the form of stilbestrol, and 1 had had an orchiectomy. In every case, histopathological study of tissue removed from the tumor prior to treatment showed active neoplasm.

The results are of interest. In 37 cases, a biopsy was obtained at least three weeks or more following the treatment. In 11 of these 37 patients, negative biopsies were obtained. Thus far, 27 of the first 50 patients treated have been alive six months or more, and have no evidence of clinical disease. Seven of the deceased cases died without evidence of clinical disease, while 8 died with evidence of disease. Table 1 gives a complete summary of these 50 cases who have been followed for six months

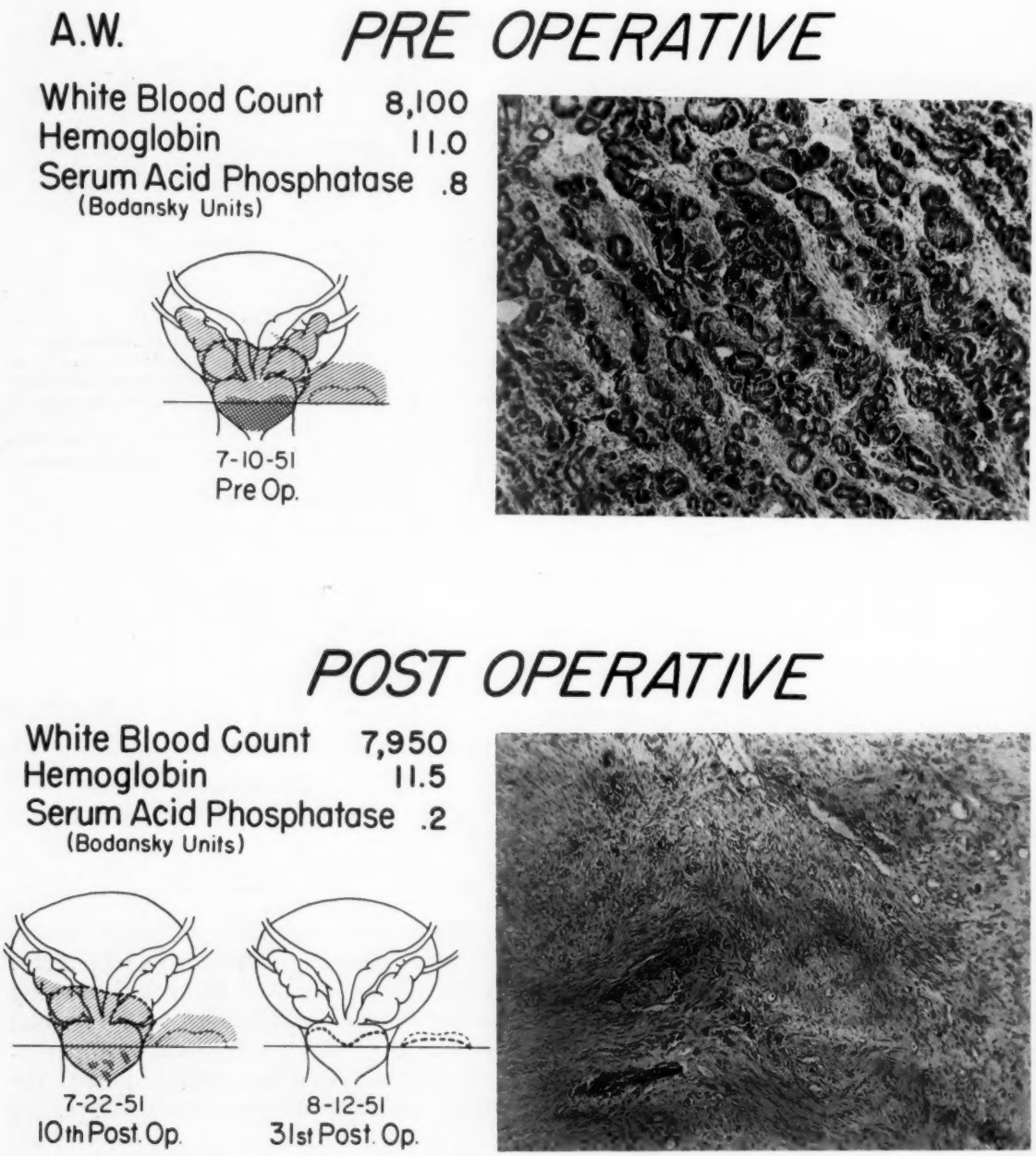

FIG. 1. This shows extent of gross involvement with the tumor and the histopathologic section before injection with Au^{198}. On the tenth postoperative day, there was considerable softening and decrease in the size of the mass. Examination on the thirty-seventh postoperative day showed almost complete disappearance of all palpable tumor while biopsy on this date reveals only questionable residual neoplasm.

or more. Figures 1 and 2 show gross and pathologic changes before and after the injection of Au^{198}.

Complications were confined almost entirely to the rectum. In the first 50 cases, there were 15 who had some sort of rectal reaction. Thirteen had tenesmus, 4 had bleeding, 3 resulted in stricture and there were 4 ulcers, 3 of which required colostomy. As an indication that careful observation and experience in this technical procedure is important is the fact that of

the next 67 cases, only 3 developed any signs of rectal difficulty.

COMMENT

We consider it fortunate that our first interstitial injection of radioactive colloidal gold was in the prostate. In selecting cases in Group 2, the lesion is still confined locally within the pelvis. The glandular capsule is intact and therefore confines practically all of the material to the injected sites. With the use of hyaluronidase,

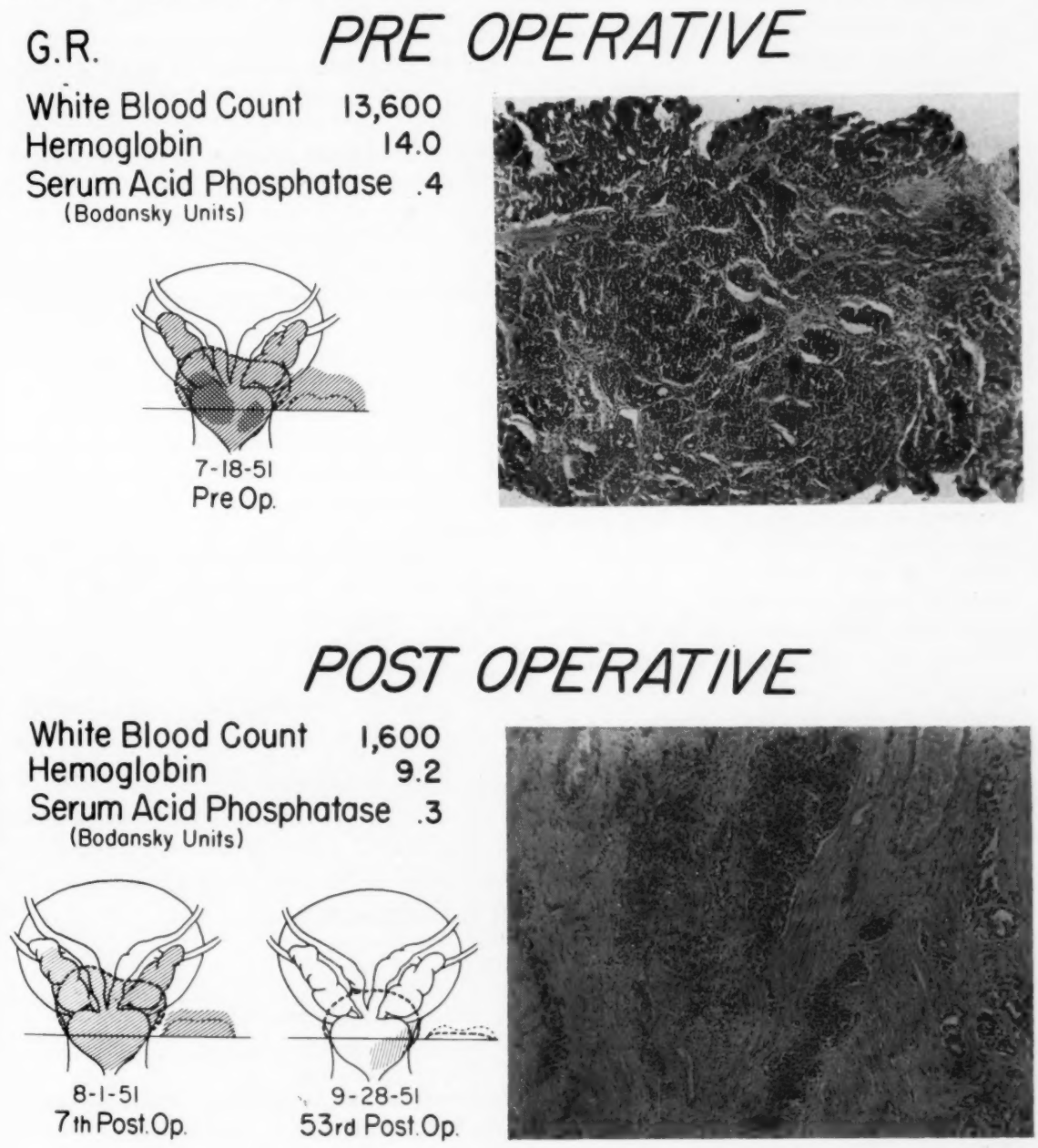

FIG. 2. Gross tumor and biopsy before treatment. Shrinkage of the mass on the seventh and fifty-third postoperative days, with persistent tumor shown in biopsy taken on latter date. Subsequent examination revealed no suspicious tumor by palpation, but no further biopsy has been taken. The patient is without clinical evidence of disease.

a small amount passes out through the lymphatics, as noted above. This is not enough to do any damage, however. It is important to select cases as carefully as possible in order that obviously hopeless cases are not included. There are already satisfactory and perhaps better methods of caring for these patients. The chief reason for utilizing this newly available material is that we have a possible means of completely destroying a malignant neoplasm where no other procedure has proved efficacious in this regard. This can be done because the radiation is mainly of the beta variety and can therefore be confined relatively easily. Also, there is a gamma component which is sufficiently strong to make the total radiation more uniform. Adjacent structures, such as rectum and bladder, are rather well protected. The fact that at least 98 per cent of all tumors showed a prompt and significant decrease in size serves to emphasize our thought that the important points to be sought are proper distribution and the correct dose. These must be worked out with meticulous care. Distribution seems to be fairly well under control by using hyaluronidase and epinephrine and injecting the solution under considerable pressure with a special rack and pinion attachment to the syringe. Dosage can only be worked out gradually by observing the reactions of the neoplasm and normal structures to varying doses in a large number of patients. As with other types of interstitial radiation, total amounts of neoplastic tissue will tend to determine the ultimate millicurie per gram ratio.

It must again be emphasized that even with careful selection of cases who are supposed to fall in Group 2, gross errors may be made and that some patients may be treated with Au^{198} mainly because they have already been operated on. Had the exact extent of the lesion been known, some of these would not have been selected for this type of treatment. It is important, therefore, to at least rule out metastatic lesions by roentgenographic examination and phosphatase determinations before selecting a patient for treatment with Au^{198}.

SUMMARY

1. We have reported on fifty cases of carcinoma of the prostate treated by the injection of Au^{198} with a follow-up of from six to seventeen months. A total of 129 cases have been treated long enough to observe that about 98 per cent show prompt and rapid decrease in the size of the palpable neoplasm.

2. Cases in Group 2 are best suited for this method of therapy.

3. Eleven of thirty-seven cases with postinjection biopsies showed no evidence of neoplasm.

4. Of the fifty cases studied in detail and observed from six to seventeen months, twenty-seven are alive without evidence of clinical disease and seven died without evidence of carcinoma. Eight are alive with clinical disease and eight died with the disease.

5. Complications are almost exclusively confined to rectal irritation. There were fifteen cases in the first fifty and three in the subsequent sixty-seven who had this complication. Four developed rectal ulcers, three of which required colostomy. Careful injection and estimation of dosage are considered the most important desiderata in this method of therapy.

6. The closest collaboration between the urologist and a radiologist well versed in isotope therapy must be assumed.

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A SIMPLIFIED INTRACAVITARY INJECTION TECHNIQUE FOR RADIOACTIVE GOLD*

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A METHOD for handling and administering radioactive colloidal gold (Au^{198}) is herein described. The method is suited to any administration performed with a single injection wherein volumes of 100 milliliters or more are permissible.

Satisfactory methods for handling radioactive colloidal gold (Au^{198}) have previously been described by Tabern[1] and Ter-Pogossian and Sherman.[2] For intracavitary injection of Au^{198} the apparatus can be simplified so that exposure from handling of radioactive material can be further reduced.

To construct the apparatus, the following equipment is needed:

1. An infusion set with a two-way stopcock interpositioned in the rubber tubing some 8 inches below the drip chamber.
2. One 30 cc. syringe.
3. Two two-way stopcocks.
4. One ½ inch 18 gauge needle.
5. One 2 inch 18 gauge needle.
6. A 4 inch length of small bore copper tubing.
7. A length of polyethylene tubing long enough to reach from the gold apparatus to the patient.
8. One 18 gauge needle of a length sufficient to gain entry into the cavity to be irradiated.
9. A lead container to hold the vial of radioactive gold.

This container is a hollow brass jacketed lead cylinder designed to fit exactly the 15 cc. bacterin vial in which the radioactive gold is shipped. This container is 3.5 inches in diameter and 3.25 inches high. This provides 1 inch of lead as a shield around the vial and will reduce the radiation intensity to something less than 1 per cent of the unshielded intensity. A disc of lead, 1 inch in thickness, provides a cover for the container and completes the enclosure of the vial.

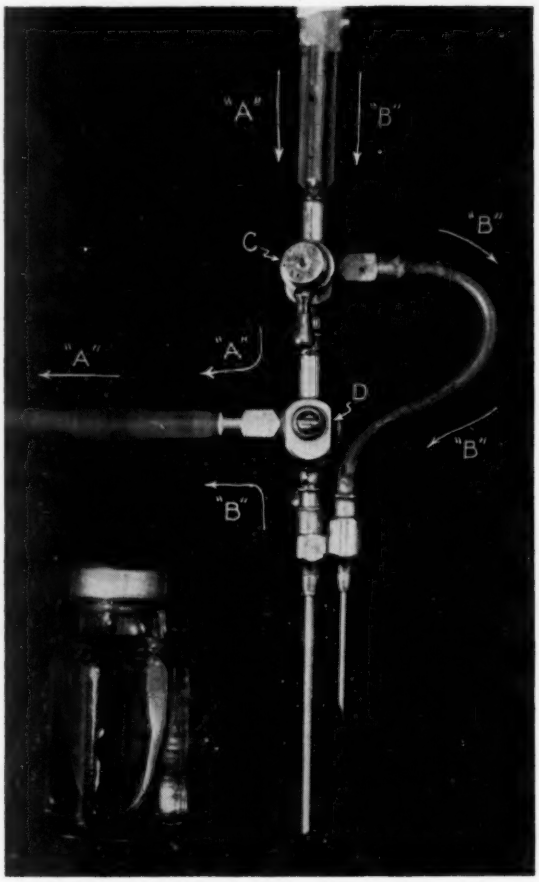

FIG. 1

The vial containing the radioactive gold is transferred from the shipping container to this special lead beaker and the remainder of the apparatus is assembled in the following manner:

The 30 cc. syringe (item 2 above) is connected to the two-way stopcock in the infusion set (item 1 above). Then items 3, 4, 5, and 6 are assembled as shown in Figure 1. The tube from the infusion set is con-

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nected to the uppermost of the two-way stopcocks and the length of polyethylene tubing (item 7) is connected to the lower stopcock.

The 2 inch 18 gauge hypodermic needle and the ½ inch 18 gauge needle are then forced simultaneously through the rubber stopper of the Abbott bacterin bottle. Then all is in readiness (Fig. 2).

The apparatus is so arranged that the infusion can be started with normal saline and thus check the patency of the equipment; then, by adjusting the two stopcocks (*C* and *D*, Fig. 1), the radioactive gold will be delivered to the patient.

With the stopcocks (*C* and *D*) set so that the outlets to the copper tube and the 2 inch 18 gauge needle are closed, the saline will follow route *A* from the reservoir to the patient, by-passing the gold. Changing the lever positions of the stopcocks *C* and *D*, will reroute the saline along route *B* into the bacterin vial containing the radioactive gold. The increased pressure thus formed in the bottle will force the gold and saline mixture up the long needle through the connecting tubing to the patient.

Additional pressure to combat any back pressure which may be encountered, or to force the last drops from the vial can be accomplished by proper manipulation of the 30 cc. syringe and its stopcock which is situated in the reservoir line between the saline reservoir and stopcock *C*.

This apparatus has been used three times in this institution to deliver doses of about 100 mc. Au^{198} and in all instances the apparatus proved to be extremely convenient to use. The best criterion for evaluating the efficiency of the delivery technique lies in the measurement of the residual radiation levels of the vial upon completion of the injection. In every instance to date, the emitted radiation of the Au^{198} vial has been 0.10 r per hour or less after delivery.

Fig. 2

The apparatus was designed for intraperitoneal injection; however, there is no reason why this arrangement should not be equally successful for intrapleural or intravenous injections.

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THE CIRCULATION OF RADIATION DAMAGED SKIN

RADIOSODIUM CLEARANCE STUDIES*†

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RADIATION damage to skin has long been a problem of paramount importance to the therapeutic radiologist. In recent years this problem has gained far wider interest following the rapid development of the military, industrial and biological applications of a variety of ionizing radiations.

This is a study of the blood flow in human skin previously damaged by roentgen rays. The results of our radiosodium clearance measurements led to the startling observation that the effective blood flow in these densely fibrotic, scarred and atrophied tissues is functionally unimpaired at any time up to ten years following roentgen therapy.

It is almost universally held that the late deleterious changes in irradiated skin are *directly* the result of the impact of ionizing radiations upon the epithelial cells, and *indirectly* the result of starvation and anoxia of these cells following radiation trauma to their blood vessels, with impairment of effective blood flow.[3,8,10,13,16,17,18,23,26–30,34]

Several investigators, notably Pullinger[19] and Ewing,[11] have expressed the opinion that the impairment of vascular supply is principally or even wholly the reason for deleterious changes in irradiated tissues. Shields Warren[28,29] asserts that while the *early* effects are caused by direct injury to the cells, the *late* changes are brought about principally by changes in the vascular bed of the corium, impoverishing the nutrient supply. Stafford L. Warren[26] states that the single greatest secondary factor in skin changes is the question of cell nutrition, as modified by cellular damage.

These opinions are based almost entirely upon the *morphological* studies of these and other investigators over the past fifty years. However, there have been no published experimental assays of the *functional* capacity of the surviving blood vessels in radiation damaged skin in the living human subject. It is the objective of the present study to present such observations of circulatory function, employing a radioisotope tracer technique.

Conventional methods for assaying the skin circulation—such as the use of the histamine wheal test, the plethysmo-

* Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

† Presented at the Thirty-fourth Annual Meeting, American Radium Society, Chicago, Ill., June 8–10, 1952.

FIG. 1

(*A*) Case 32. Marked atrophy and telangiectasia nine years after 3,990 r to skin in twenty-seven days for advanced laryngeal cancer.

(*B*) Case 33. Extensive scarring and induration twenty-two months after 6340 r to skin in twenty-seven days for inoperable male breast cancer. Note full thickness graft in center of lesion where radiation ulcer persisted.

(*C*) Case 28. Scarring and massive induration more than five years after 4,200 r to skin in thirty-seven days (1 mv.) for carcinoma of testicle.

(*D*) Case 31. Massive induration and shrinkage $5\frac{1}{2}$ years after 5,000 r to skin in 72 days (1 mv.) for carcinoma of testicle.

(*E*) Case 6. Hyperintensive second degree skin reaction after 13,284 r to open areas in grid in forty-six days for carcinoma of pharynx.

(*F*) Case 6. Note grid pattern five months later with pale areas of severely atrophic skin.

Fig. 1

FIG. 2

graph, the saline wheal test, and the visualization of fluorescein dye—are generally regarded as not entirely satisfactory since they do not reflect relatively small changes in blood flow. It was Kety[14] who first suggested that if a diffusible tracer substance such as the Na^{24} ion (as NaCl) were injected into any specific living tissue under consideration, then its clearance from such tissue would depend upon and be a reliable measure of the efficiency of the local tissue circulation.

CLINICAL MATERIAL

Our clinical material (see Table 1) comprises 37 individuals presenting one or more areas of skin with severe residual radiation damage following the application of roentgen therapy in the past ten years for a variety of malignant lesions. In these cases, there was observed marked atrophy, scarring, induration, contracture, and telangiectasis (Fig. 1 and 2). In 9 cases healing was delayed for several weeks or months following radiotherapy (Fig. 1 and 2).

The patients in our study had been treated with a wide variety of roentgen therapy techniques representing nearly the entire spectrum of therapeutic roentgen radiations. The kilovoltage ranged from 45 kv. to 2,000 kv., and the quality from half-value layer 0.3 mm. Al to half-value layer 7.1 mm. Pb. The skin dose ranged from 3,600 to 20,400 roentgens delivered in overall time periods varying from thirty seconds to 100 days. In nearly one-half the patients, the skin dose exceeded 6,000 r. In 7 individuals (Cases 1, 3, 6, 16, 22, 33 and 34), the Marks grid was employed for a total skin dose of 10,360 to 20,400 r (measured in the open areas). The reported skin dose and treatment time in 6 subjects (Cases 1, 16, 20, 23, 29 and 37) represent a summation of two courses given within a relatively short time of each other.

In an attempt to correlate experimental assays of circulatory function with histopathologic evidence of vascular damage, skin biopsies were secured from 15 lesions presenting the most severe degree of radiation damage. A sharp annular punch 6 mm. in diameter was employed to obtain full-thickness skin specimens. In the Marks grid cases, the biopsies were secured from skin areas exposed through the openings in the grid.

METHOD

The patient was placed at rest in a temperature controlled room for ten to fifteen minutes prior to the clearance study. The technique was carefully explained to the individual in order to allay any possible apprehension regarding the injection or the counting procedure. However, in no instance was any evidence of fear or apprehension encountered. After the rest period, 0.05 cc. of isotonic solution containing 1 to 3 microcuries of radioactive sodium (Na^{24}) was injected intradermally in the irradiated skin, employing a 26 gauge needle and raising a tiny bleb. A similar injection, as a control, was made in the normal skin of the identical region on the opposite side of the body. All patients were tested under identical physiological and environmental

←

FIG. 2

(*A*) Case 36. Striking atrophy 3½ months after 6,560 r to skin in thirty-six days for nasopharyngeal cancer. Several weeks elapsed before healing occurred.

(*B*) Case 36. Histological section from same case illustrating intact lumen of arterial vessel showing only slight evidence of damage. Marked damage to epidermis, corium and adnexa was noted in the *entire* section.

(*C*) Case 18. Severe contracture, induration and scar after 3,600 r to skin in thirty-five days (1 mv.) for carcinoma of testicle.

(*D*) Case 18. Histological section from same case illustrating numerous dilated capillaries and endothelial lakes despite evidence of much damage to epithelium and corium.

(*E*) Case 10. Atrophy, scarring and contracture ten years after 5,836 r to skin of neck for advanced laryngeal carcinoma.

(*F*) Case 10. Portion of histological section from same case showing only partly damaged functioning arterioles. The remainder of the section showed extensive and very severe damage to epidermis and corium.

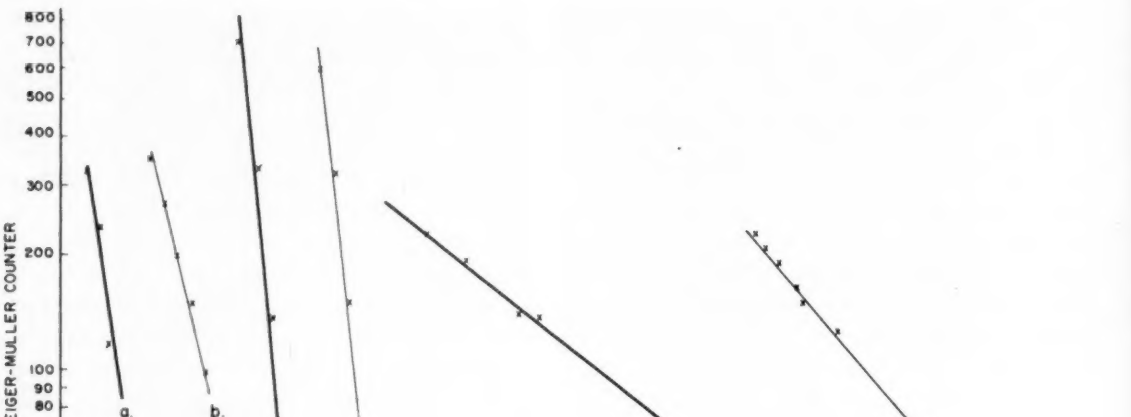

CHART I

I* = Physiological state.
II = Following histamine iontophoresis.
III = Following adrenalin.

conditions at the same time of day, with room temperature carefully controlled. A Geiger-Müller counter with thin mica window was placed in a fixed position over the site of intradermal injection. The counts were recorded at one minute intervals for twenty minutes and plotted against time on a semilogarithmic graph. The value of k (the clearance constant), was determined from the slope of the resulting straight line (Chart 1).

Inasmuch as the injection is very superficial, the counter detects the radiation of beta particles and, with lesser efficiency, the gamma rays from the injected radiosodium, thus making it possible to use 0.03–0.05 cc. of solution, less than one-tenth of the dose employed by Kety.[14,15]

RESULTS

A total of 74 radiosodium clearance studies was performed in 37 subjects (Table 1). In each subject a clearance test was made in the irradiated skin and in normal skin in a contralateral area, the latter being considered as a control. We have expressed in percentage the difference between the rate of clearance in the irradiated skin and in the control (Table 1).

In 4 subjects, there was no difference in the clearance rates, comparing the damaged skin and the normal skin in the same patient. In 9 subjects, the clearance rate of the irradiated skin was slower than its control. In 24 other cases the clearance rate of the irradiated skin was faster than the control rate. If we consider that a ± 20 per cent error is inherent in the technique (Wisham, Yalow and Freund[31]), there is only 1 subject with an irradiated skin clearance rate slower than the control and 12 subjects whose irradiated skin clearance rate was faster than the control (Table 1).

In 4 subjects who received histamine by

TABLE I

SUMMARY OF CLINICAL AND LABORATORY DATA

Case No.	Cancer Treated	Skin Portal	Size of Portal cm.	Time Since Irriadiation	Skin Dose (r)	Treatment Time days	Kv.	Filter (mm.)	Half-Value Layer (mm.)	Half-Life (min.)		Clearance Factor (k)		Per Cent Var.
										Lesion	Control	Lesion	Control	
1	Kidney	Back, lumbar	12×12	3 mo.	12,295*	49	260	Thoraeus	2.5 Cu	5.25	5.50	0.1321	0.1240	+ 8.3
2	Bronchus	Chest, ant.	15×24	10 wk.	5,750	28	2000	4.0 Pb	7.0 Pb	5.25	4.75	0.1321	0.1438	− 8.3
3	Esophagus	Chest, ant.	6×12	3.5 wk.	17,270*	39	260	0.5 Cu	1.5 Cu	5.75	6.50	0.1206	0.1026	+17.5
4	Skin	Face, right	3.5 diam.	7 yr.	4,280	10	100	None	2.0 Al	3.00	3.50	0.2278	0.1954	+16.5
5	Mouth	Neck, left	10×15	4 wk.	4,485	30	260	Thoraeus	2.5 Cu	1.75	2.00	0.3920	0.3410	+11.5
6	Pharynx	Neck, post.	10×15	5 mo.	13,284*	46	260	0.5 Cu	1.5 Cu	2.50	2.75	0.2755	0.2480	+10.5
7	Hodgkin's disease	Abdomen, ant.	10×12	2 yr.	4,480	21	200	2.0 Cu	1.9 Cu	3.00	4.50	0.2278	0.1542	+42.0
8	Nasopharynx	Neck, left	10×15	5.5 yr.	4,500	34	1000	3.0 Pb	3.6 Pb	2.50	3.25	0.2755	0.2102	+13.1
9	Skin	Face, left	5×7	13 mo.	7,560	16	200	0.5 Cu	1.0 Cu	3.75	3.50	0.1850	0.1975	− 6.2
10	Larynx	Neck, right	8.0 diam.	10 yr.	5,836	34	200	2.0 Cu	1.8 Cu	3.75	4.75	0.1848	0.1438	+22.8
11	Parotid	Face, right	6×10	8 yr.	6,000	34	250	1.5 Cu	1.5 Cu	4.75	4.25	0.1438	0.1634	−11.9
12	Skin	Back, right	1.5 diam.	2 yr.	7,000	0.5 m	45	None	0.3 Al	4.50	4.50	0.1540	0.1540	0
13	Larynx	Neck, right	4×5	9 mo.	6,634	30	250	Thoraeus	2.5 Cu	2.25	2.25	0.3035	0.3035	0
14	Tongue	Face, right	11×11	15 mo.	7,712	59	250	Thoraeus	1.5 Cu	4.25	3.75	0.1634	0.1848	−11.8
15	Tonsil	Face, right	10×10	2.7 yr.	6,238	32	200	Thoraeus	2.0 Cu	1.75	2.00	0.3920	0.3410	+15.0
16	Buccal	Face, right	6×15	20 mo.	20,170*	69	220	0.5 Cu	1.0 Cu	3.50	4.75	0.1982	0.1438	+37.5
17	Tongue	Face, right	9×10	8 mo.	6,019	34	220	Thoraeus	2.1 Cu	4.50	4.25	0.1540	0.1634	− 5.6
18	Testicle	Abdomen, ant.	10×15	5.7 yr.	3,600	35	1000	3.0 Pb	3.6 Pb	10.75	11.25	0.0644	0.0616	+45.0
19	Larynx	Neck, right	10×16	6 mo.	5,440	24	200	5.0 Cu	1.0 Cu	3.00	3.50	0.2278	0.1954	+15.4
20	Leukemia	Abdomen, ant.	10×15	7.5 yr.	7,265	100	400	Thoraeus	4.7 Cu	5.25	7.25	0.1321	0.0946	+39.0
21	Cervix	Back, lumbar	10×15	3 mo.	4,758	14	400	Thoraeus	4.7 Cu	2.75	2.75	0.2480	0.2480	0
22	Bronchus	Chest, post.	10×10	4 wk.	18,000*	42	260	0.5 Cu	1.5 Cu	8.25	8.50	0.0837	0.0802	+ 5.5
23	Bronchus	Chest, ant.	8×10	6 mo.	20,400*	126	400	Thoraeus	4.7 Cu	2.50	5.00	0.2730	0.1366	−100.0
24	Buccal	Neck, right	8×9	7 wk.	6,380	28	400	Thoraeus	4.7 Cu	3.25	4.75	0.2100	0.1438	+46.0
25	Vallecula	Neck, right	8.0 diam.	20 mo.	5,280	28	220	Thoraeus	2.1 Cu	1.50	3.00	0.4550	0.2278	−100.0
26	Tonsil	Neck, left	7.0 diam.	6 mo.	5,550	27	400	Thoraeus	4.7 Cu	5.00	4.75	0.1366	0.1438	− 5.0
27	Spine	Back, lumbar	8×15	22 mo.	5,600	28	140	0.25 Cu	0.5 Cu	2.25	2.25	0.3035	0.3035	0
28	Testicle	Back, lumbar	10×15	5.3 yr.	4,200	37	1000	3.0 Pb	3.6 Pb	6.75	6.50	0.1010	0.1050	− 3.2
29	Bronchus	Neck, left	8×8	1.5 yr.	9,270	40	200	1.0 Cu	1.5 Cu	3.70	4.75	0.1851	0.1432	+29.0
30	Pleura	Chest, left	15×23	3.5 wk.	4,500	35	2000	4.0 Pb	7.1 Pb	3.00	2.00	0.2278	0.3410	−33.0
31	Testicle	Abdomen, ant.	10×15	5.6 yr.	5,000	72	1000	3.0 Pb	3.6 Pb	7.00	6.25	0.0972	0.1092	+11.0
32	Larynx	Neck, right	8×10	9.2 yr.	3,990	27	200	0.5 Cu	0.9 Cu	6.00	8.75	0.1138	0.0755	+50.0
33	Male breast	Chest, left	12×12	22 mo.	6,345	27	260	Thoraeus	2.5 Cu	3.00	3.50	0.2278	0.1954	+15.0
34	Thyroid	Neck, left	12×12	15 mo.	10,360*	36	260	Thoraeus	0.5 Cu	5.50	5.75	0.1240	0.1206	+ 2.8
35	Parotid	Face, right	10×10	9 yr.	6,800	65	200	2.0 Cu	1.8 Cu	2.00	2.50	0.3410	0.2730	+25.0
36	Nasopharynx	Face, left	8×10	3.5 mo.	6,560	36	260	Thoraeus	2.5 Cu	1.75	2.75	0.3920	0.2480	+58.0
37	Tongue	Face, right	10×10	16 mo.	10,875	63	400	Thoraeus	4.7 Cu	2.50	3.50	0.2730	0.1954	+38.5

* Treated through Marks grid.

iontophoresis (to the irradiated skin and a control site), the clearance rate was increased in all but one area (Table II, Chart 1). The increases in clearance rate due to histamine were approximately equal in normal and radiation damaged skin.

The 3 patients who received adrenalin by iontophoresis showed a marked slowing of clearance rate in both irradiated skin and the control site. These rates were approximately equal in pathological and normal areas. Two patients were given injections of Na^{24} solution mixed with adrenalin in irradiated skin and the control site. There resulted a marked slowing of clearance rate as compared with the clearance rate in the physiological state and the responses were the same in both normal and pathological skin areas.

In 7 subjects in this study, temperature readings were taken in the irradiated skin and a control contralateral area. In 2 subjects, the temperature of the irradiated skin was greater than the normal skin. In 2 subjects the temperature readings of the irradiated skin were exactly the same, and in 3 subjects the temperature of the irradiated skin was less than that of the normal skin. In no case was there a difference in temperature readings from the two sites, pathological and control, exceeding 1 degree Fahrenheit (Table III).

Histopathological examination of biopsy specimens from the irradiated skin in 15

TABLE II

RESULTS OF HISTAMINE AND ADRENALIN TESTS

Case No.	Test Condition	Clearance Constant (k)	
		Lesion	Control
1	Physiological	0.1321	0.1240
	Histamine iontophoresis	0.3410	0.2102
	Adrenalin iontophoresis	0.0154	0.0462
3	Physiological	0.1206	0.1026
	Histamine iontophoresis	0.2480	0.1634
5	Physiological	0.3920	0.3410
	Histamine iontophoresis	0.5521	0.4620
	Adrenalin iontophoresis	0.0865	0.1053
22	Physiological	0.0837	0.0802
	Adrenalin iontophoresis	0.0718	0.0325
23	Physiological	0.2730	0.1366
	Histamine iontophoresis	0.2480	0.2278
24	Physiological	0.2100	0.1438
	Adrenalin with Na^{24}	0.0239	0.0184
33	Physiological	0.2278	0.1954
	Adrenalin with Na^{24}	0.0285	0.0273

patients revealed typical severe radiation changes involving the epidermis, corium, adnexa and vascular structures. They were identical with those already described in intimate detail by numerous authors over the past fifty years. In the epidermis was noted marked atrophy, occasionally with ulcerations. A few lesions showed marked acanthosis, hyperkeratosis, and occasional parakeratosis. In the stroma there was noted considerable densification and hyalinization of the collagen fibers. Giant radiation fibroblasts were often encountered. The elastic fibers showed a marked degree of hyperelastosis, with some fragmentation and disorganization. The hair follicles and sebaceous glands presented evidence of severe injury and were often entirely obliterated. Even the sweat glands, recognized as the most resistant of the dermal appendages, showed evidence of severe radiation injury with shrinkage and atrophy.

TABLE III

RESULTS OF THERMOCOUPLE TESTS

Case No.	Site	Temperature (F.)	
		Lesion	Control
17	Face, right	97.5°	98.5°
20	Abdomen, ant.	100.5°	101.0°
28	Back	100.0°	100.0°
31	Abdomen, ant.	99.0°	99.5°
33	Chest, left	98.0°	97.0°
34	Neck left.	101.0°	100.5°
36	Face, left	98.0°	98.0°

Vascular damage of varying severity was noted in every section studied. It was characterized by endothelial proliferation, hyalinization of the media, and varying degrees of narrowing of the arterial lumen. Marked tortuosity was suggested in several of the sections (Fig. 2, *F*). Recanalization was occasionally evident. Dilated telangiectatic endothelial vessels were frequently noted. Despite widespread evidence of vascular injury, one could often see apparently well functioning dilated capillaries, narrowed but patent arterioles and arteries, and even entirely unharmed vessels (Fig. 2, *B*). These findings are similar to those already reported by numerous authors.[3,7,8,10,11,13,16,17,18,23,25,26–30,34]

The correlation of these radiation changes with dose, time and quality of roentgen radiation will be considered in detail in a later communication.

DISCUSSION

Late deleterious changes in radiation damaged skin have almost universally been described as the result of both direct and indirect action of irradiation. The most important indirect factor has been assumed to be impaired circulatory function secondary to obliterative endarteritis involving the local blood vessels. This assumption has been based on extensive studies of the *morphology* of irradiated skin and other tissues over the past fifty years.[3,8,10,11,13,16,17,18,23,26–30,34] While such an assumption may appear quite reasonable, we have been able to find no assays of circulatory *function* in irradiated skin to support this assumption. Indeed, the radioisotope tracer technique appeared to offer for the first time an accurate index of the

local circulation and to provide a real test of the validity of the assumption.

Our findings are not in agreement with the universally accepted theory of impaired vascular function as one of the important factors in the late pathological changes in irradiated skin. Therefore, it is necessary that we first establish the validity of the radiosodium clearance technique.

The basis for this test was first suggested by Kety[14] and by Cooper and co-workers.[5] They postulated that since Na^{24} is a small, freely diffusible ion, it would disappear from the site of injection at a constant rate if the rate of blood flow was constant. They also said that this rate of disappearance would be one of constant exponential and, if plotted on a semilogarithmic graph against time, would be a straight line.

Kety,[15] Elkin and co-workers,[9] Stone and Miller,[22] and Wisham Yalow and Freund,[31] all demonstrated that the rate of disappearance of radiosodium from *muscle* tissue offered a superior type of assay of the status of the regional circulation of the extremities.

Similar results were achieved by Wisham and Yalow,[32] using the *subcutaneous* tissues of the human leg. Conway, Roswit, Stark and Yalow[4] applied a radiosodium clearance technique to the study of circulation of *skin* by testing tubed skin pedicles during the progress of reconstructive plastic surgery in veterans. These authors sought a critical test for determination of the optimum time at which division and migration of the skin pedicles was most likely to be successful. They were convinced that this method provided an objective, quantitative and entirely accurate index of the efficiency of the local skin circulation. Similar observations were reported about the same time by Braithwaite, Farmer and Herbert,[2] and by Barrow, Veall and Arnott.[1] It was shown by Wisham and his co-workers[31,32] that the clearance rate of Na^{24} from muscle tissue could be reproduced over long time periods within a relatively narrow range.

It may be suggested that sodium disappears from the site of injection by diffusion along tissue planes and clearance is therefore not dependent on blood flow. That this does not occur has been concluded by Stone and Miller,[22] and by us from unpublished data.[33] If a Geiger-Müller counter with $\frac{3}{4}$ inch opening is placed just laterally adjacent to the site of a skin injection of Na^{24} so that not part of it is over the site of injection, there will be no radioactivity recorded.

It may be suspected that some of the sodium ion is picked up by the lymph vessels. Stone and Miller,[22] in an ingenious experiment, showed that this mechanism does not take place to any significant degree. They cannulated the femoral vein and thoracic duct of a dog and then injected Na^{24} in a muscle of the dog's leg. Blood and lymph were then collected until the Na^{24} had disappeared from the site of injection. They found that 98.5 per cent of the radioactive material was present in the blood collected from the femoral vein and that there was practically no radioactive material in the lymph collected from the thoracic duct.

Since it has been shown that the sodium ion does not diffuse along tissue planes and is not carried away through lymph channels, one must conclude that it is carried away through the blood vessels alone.

There are substantial data already available indicating that the technique employed truly measures the blood flow. For example, it has been observed (Table 1) that the clearance rate from the skin of the face and neck in man is more rapid than that found in the skin of the abdomen and extremities. This is in agreement with the observations of plastic surgeons that these areas are more richly supplied with blood vessels than the skin of the abdomen and extremities. Furthermore, the clearance rate from subcutaneous tissues, when compared to the clearance rate from skin and muscle in man, appears to be much slower.[31,32] It is a well known fact that muscle and skin have a greater number of blood vessels than subcutaneous tissues.

Many experiments have been performed designed to change the rate of blood flow and at the same time determine the Na^{24} clearance rate. The simplest experiment is to place a tourniquet about the part tested and occlude the artery and vein, thus preventing blood flow to the part.[15] In every such case, the clearance of Na^{24} has been slowed as expected; further, when the tourniquet is released, the clearance has been greater than the rate prior to the use of the tourniquet. This is a result of reactive hyperemia.[20] As another example, exercise has been shown to increase the rate of clearance in muscle to a great extent, and it is well known that blood flow is increased during exercise.[31] Reflex heating of the abdomen increases the rate of clearance in the extremities as expected.[20]

We have attempted to change the blood flow in the normal and radiation damaged skin of several subjects by giving histamine and adrenalin by iontophoresis. Histamine causes dilation of blood vessels and a resultant increase in blood flow; this was shown by an increase in the rate of clearance of Na^{24} compared to previously determined clearance rates from the same site. The effect of adrenalin is to constrict the blood vessels, resulting in a slowing of blood flow. This was demonstrated by a decrease in the clearance rate of Na^{24}. If adrenalin is injected into the skin mixed with the Na^{24} solution, the result is a very slow clearance rate (Table II).

We have done experiments measuring the clearance rates from skin in which the sympathetic nerve was interrupted by sympathectomy (unpublished data). In comparing the clearance rates in the same patient prior to surgery, the rate appears to be increased considerably after surgery. This is obviously the direct result of dilation of vessels after sympathectomy with increase of blood flow in the skin.[33] A more detailed report on these experiments will be made in a later communication.

Studies performed in skin prior to and during the course of roentgen therapy have shown an increased clearance rate as the erythema became evident, and a return of the clearance rate toward normal values as the erythema subsided.[33] Further studies in this field will be reported in a later communication.

In all histological sections there was noted classical evidence of damage to blood vessels of varying severity. However, there were always observed numerous relatively undamaged dilated capillaries, in some instances appearing in the form of huge endothelial lakes simulating cavernous hemangioma. This is a common observation in irradiated human skin and was first described by Wolbach in 1909.[34] Ewing,[11] Wolbach[34] and Warren[28] believe that new capillaries often develop in the subepidermal area and grow actively. Although most of the larger vessels showed partial narrowing of the lumens, they were generally patent and contained blood cells. In fact, some arterioles showed no signs of radiation damage whatsoever. Shields Warren[27] noted that even in severely damaged areas some normal blood vessels were seen. In fact, he further states that the larger the vessels, the greater is the chance for repair and maintenance of patency. This observation is well supported by Davidoff *et al.*[7] and Wolbach.[34]

The undamaged and the new capillaries must dilate because there are fewer such channels to handle incoming blood. Therefore, each surviving capillary carries a greater volume of blood than the capillaries of normal tissue. As a result, the total volume of blood flowing through the capillaries of irradiated skin may approach the total volume of blood flowing through the same volume of normal skin.

Damage and destruction of elastic tissue is nearly always observed. This results in shrinkage of the bulk of irradiated tissue. Therapeutic radiologists are aware of the marked contraction of the treated skin portals observed over a period of years following radiation therapy. An excellent example is illustrated in Figure 1. Since less tissue is present, the relation between volume of blood flow and unit amount of

tissue in normal and irradiated skin may be unchanged although a smaller total volume of blood may flow through the surviving capillaries.

Free and even excessive bleeding from irradiated tissues during operative procedures is commonly encountered by plastic surgeons. It was first mentioned by Porter and White[18] as early as 1907. This suggests an adequate circulation. If there were severe obstructions to blood flow, such tissue should not bleed freely. In disease processes such as thromboangiitis obliterans where capillaries are markedly occluded, there is very little bleeding during surgery of the involved extremities.

We have measured the skin temperatures of radiation damaged skin with a thermocouple and compared them with the temperatures of the contralateral area of normal skin (Table III). In every case, there was practically no difference in the reading. This supports our findings of normal clearance rates in radiation damaged skin. If the blood flow to radiation damaged skin was considerably less than normal, one would expect the temperature of the area to be less than that of normal skin.

It may be suggested by some that the *pallor* of irradiated skin indicates impaired circulation. The densification and hyalinization of the subepidermal connective tissue obstructs the visualization of the capillaries from above, and this accounts for the pallor, despite the maintenance of adequate circulation.

If one accepts the fact that the radiosodium clearance determination is a valid test of effective blood flow, one must conclude that the effective blood flow of radiation damaged skin, within presently employed therapeutic dose levels, is within normal limits. It follows that the late deleterious changes noted in irradiated skin are not the result of impairment of vascular supply, but due to *direct* radiation injury to the cells of the epidermis and supporting tissues. Studies are already in progress to determine whether the same observations obtain in radiation responsive human neoplasms during and after a course of radical roentgen therapy.

SUMMARY

1. The radiosodium clearance rate was determined in each of 37 subjects with severe radiation damaged skin and in a control contralateral area in the same patient.

2. The clearance rate from the radiation damaged skin and from the control skin was similar.

3. These findings indicate that the effective blood flow in these lesions is within normal limits, despite histopathological evidence of varying degrees of vascular damage. This further suggests that vascular damage observed plays no role in the late deleterious changes in human skin following therapeutic irradiation.

4. Substantial evidence for the validity of the radiosodium clearance technique as a qualitative index of effective blood flow has been reviewed, extended and discussed.

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GRID DEPTH DOSE INVESTIGATION FOR 200 AND 400 KILOVOLTS AT THE CENTER AND EDGE OF THE FIELD

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THIS report, a continuation of a previous study[1] on depth doses with a 40 per cent transmission grid, gives the methods and the results of measurement at the center and edge of the field for 200 and 400 kilovolt radiation.

METHOD

400 kv. A cone with a grid was designed for use with a light localizer on the 400 kv. machine. This arrangement permitted the visualization of the field, round light spots surrounded by darker interspaces on a masonite presdwood phantom. This cone is described in the preceding issue of this JOURNAL.[2] The grid could be moved by means of a rack and pinion in any direction in the plane of the base, thus permitting the desired positioning. The grid was made of a lead sheet 5 mm. thick fastened to a sheet of aluminum 2 mm. thick to absorb the characteristic radiation of the lead and to give it rigidity. The grid material transmitted 1.6 per cent of the incident beam whose half-value layer was 3.3 mm. Cu. The holes had a diameter of 1 cm. and a distance of 1.4 cm. between their centers.

Before the investigation was begun the light field was adjusted to coincide with the roentgen-ray field.

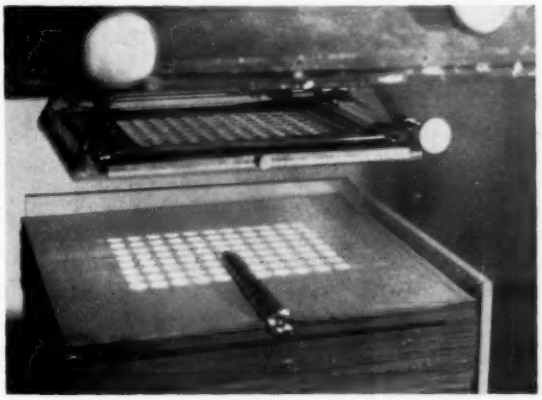

FIG. 1. Set-up for measuring depth dose with a movable grid cone and light localizer on the 400 kv. machine.

FIG. 2. Set-up for measuring depth dose at edge of field with the 200 kv. machine.

The set-up is shown in Figure 1. A Victoreen 250 r chamber was used because its measuring volume was sufficiently small to be placed in the radiation coming through a hole indicated by a light spot, or to be completely covered by the covered part of the grid between holes indicated by the shadowed portion. The size of the field was changed by means of a movable diaphragm. The positioning was done both for the center of the beam and for the edge of the beam with the roentgen tube at the distance from the phantom in which measurements were to be made. This precaution was especially necessary

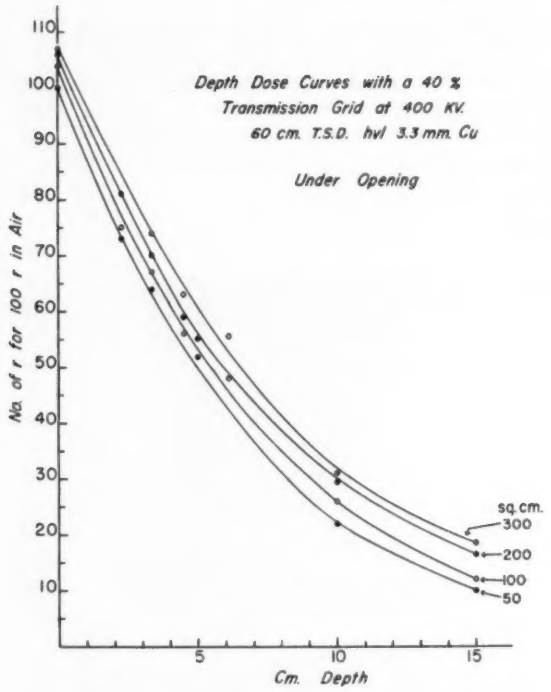

FIG. 3. Depth dose curves for center of beam under opening with a 40 per cent transmission grid, at 400 kv., 60 cm. target-skin distance, half-value layer 3.3 mm. Cu.

for the edge of the field because, as the tube is moved, the size of the light field and roentgen-ray field changes and the light spots shift.

To make measurements at the edge of the field, the holder with the ionization chamber was surrounded by strips of presdwood made by cutting a slab of three pegged boards into narrow pieces.

200 kv. Edge of Field. The set-up for the 200 kv. machine is shown in Figure 2. The edge of the plastic guide on the chamber boards was used together with the graph paper on the board underneath it to measure the displacement of the chamber from the center. Here, as in the case for the center of the beam,[1] films were taken at each depth to make certain that the chamber was either underneath a hole or underneath a covered area before measurements were begun. The size of the field was varied for each chamber position.

The grid used for the 200 kv. roentgen rays was made of lead rubber 4 mm. thick and had a lead equivalent of 1.3 mm. The lead rubber transmitted less than 1 per cent of the incident beam.

RESULTS

400 kv. The results of depth dose measurements for the center of the beam under the opening at 60 cm. target-skin distance for 400 kv. roentgen rays with a half-value layer of 3.3 mm. Cu are given in the curves of Figure 3 for field sizes ranging from 50 sq. cm. to 300 sq. cm. The curves for the depth doses under the covered portion are shown in Figure 4.

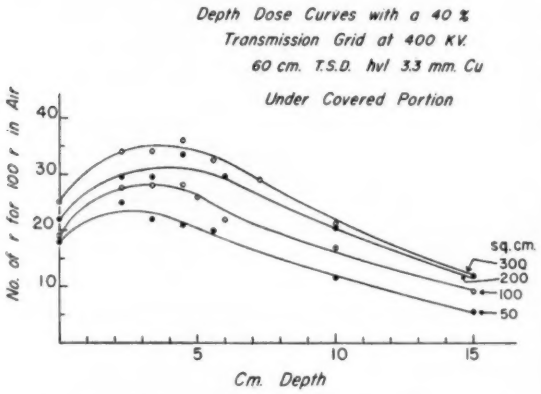

FIG. 4. Depth dose curves for center of beam under covered portion with a 40 per cent transmission grid, at 400 kv., 60 cm. target-skin distance, half-value layer 3.3 mm. Cu.

The data from these curves are tabulated for depths from the surface to 15 cm. in Table I. The average obtained by weighing the depth dose under the opening as 0.4 and that under the covered portions as 0.6 is also included.

In Table II the depth dose at the edge of the field, but within the confines of the direct beam, is compared with that at the center of the field for different field sizes: (1) for the radiation coming through the hole, and (2) for the radiation under the covered portion. In the column headed Cm. from cen. are listed the distances which the chamber was displaced to the left of the center. In the last column for each size of field headed % diff./c. beam is listed the difference between the number of r at the center and at the edge of the field expressed as a per cent of the central beam. In general, the larger the field, the larger the percentage difference.

TABLE I

DEPTH DOSE AT CENTER OF FIELD FOR 400 KV. ROENTGEN RAYS, H.V.L. 3.3 MM. Cu AT 60 CM. T.S.D.; 40 PER CENT TRANSMISSION GRID, R FOR 100 R IN AIR

Depth in cm.	Area of Field in Square Centimeters			
	50	100	200	300
	(a) Under Opening			
0	100.0	104.0	106.0	107.0
1	87.5	91.5	94.5	97.0
2	76.0	80.0	84.0	87.5
3	66.0	70.0	74.0	77.5
4	57.5	61.0	64.5	69.0
5	50.0	53.0	56.0	60.5
6	43.0	46.5	49.5	53.5
7	36.5	40.0	44.0	47.0
8	31.0	35.0	38.5	41.5
9	26.0	30.0	34.0	36.0
10	22.0	26.0	30.0	32.0
11	19.0	22.5	26.8	28.5
12	16.6	19.5	23.8	25.5
13	14.3	16.8	21.2	23.0
14	12.0	14.4	18.8	20.5
15	10.0	12.0	16.5	18.5
	(b) Under Covered Portion			...
0	17.5	19.0	22.2	25.0
1	21.5	24.5	26.5	30.5
2	23.0	27.0	29.5	33.5
3	23.5	28.0	30.5	35.0
4	22.5	28.0	31.0	35.0
5	20.5	26.5	30.5	34.0
6	18.5	23.5	29.0	32.5
7	16.5	21.0	27.0	29.5
8	15.0	19.5	24.5	27.0
9	13.5	17.5	22.5	24.5
10	12.0	16.0	20.5	22.0
11	10.5	14.5	18.5	19.7
12	9.2	13.2	16.5	17.5
13	8.0	12.0	15.0	15.6
14	6.6	10.5	13.0	14.0
15	5.5	9.5	11.5	12.0
	(c) Average			
0	50.5	53.0	55.7	57.8
1	47.9	51.3	53.7	57.1
2	44.2	48.2	51.3	55.1
3	40.5	44.8	47.9	52.0
4	36.5	41.2	44.4	48.6
5	32.3	37.1	40.7	44.6
6	28.3	32.7	37.2	40.9
7	24.5	28.6	33.8	36.5
8	21.4	25.7	30.1	32.8
9	18.5	22.5	27.1	29.1
10	16.0	20.0	24.3	26.0
11	13.9	17.7	21.8	23.2
12	12.2	15.7	19.3	20.7
13	10.5	13.9	17.5	18.6
14	8.8	12.1	15.3	16.6
15	7.3	10.5	13.5	14.6

Depth dose in r for 100 r in air, at the center of the field for 400 kv. roentgen rays, half-value layer 3.3 mm. Cu, at 60 cm. target-skin distance, with a 40 per cent transmission grid, for different field areas (*a*) under opening, (*b*) under covered portion, (*c*) average.

Under the openings the difference is greatest at the surface. It is approximately 10 per cent for field sizes up to 200 sq. cm. For a 300 sq. cm. field it is approximately 20 per cent.

The scattered radiation under the covered portion at the edge of the field remains the same for a depth of 6 to 7 cm. Since at the center this radiation increases over the surface value to a depth of 4 to 5 cm., the greatest difference between the edge and the center occurs at this depth as is seen in Table II at a depth of 5 cm. This difference at a depth of 5 cm. ranges from 20 per cent for the 50 sq. cm. field to 45 per cent for a 300 sq. cm. field. Because of this large fall off at the edge, the field irradiated should be sufficiently large to include the growing edge of the tumor well within the geometric beam.

200 kv. In Table III a comparison of the depth dose at the edge of the field for 200 kv. roentgen rays, half-value layer 0.94 mm. Cu, at a target-skin distance of 50 cm. is made with that at the center of the field. Here, as in the case of 400 kv.,

TABLE II

400 kv.

COMPARISON OF DEPTH DOSE AT EDGE OF FIELD WITH THAT AT CENTER OF FIELD; R FOR 100 R IN AIR, 40 PER CENT TRANSMISSION GRID, T.S.D. 60 CM. 400 KV., H.V.L. 3.3 MM. Cu

Depth in cm.	Area in sq. cm.															
	50				100				200				300			
	Cm. from cen.	r edge	r cen. beam	% diff. c. beam	Cm. from cen.	r edge	r cen. beam	% diff. c. beam	Cm. from cen.	r edge	r cen. beam	% diff. c. beam	Cm. from cen.	r edge	r cen. beam	% diff. c. beam
								(a) Under Opening								
0	L.3	92.0	100.0	8	L.4.5	92.0	104.0	12	L.6.5	94.0	106.0	11	L.8	83.0	107.0	22
2	L.3	71.5	76.0	6	L.4.5	72.0	80.0	10	L.6.5	72.5	84.0	14	L.8	67.5	87.5	23
5	L.3	49.0	50.0	2	L.4.5	50.0	53.0	6	L.6.5	50.0	56.0	11	L.8	45.5	60.5	25
10	L.3	21.0	22.0	5	L.4.5	24.0	26.0	8	L.6.5	26.5	30.0	12	L.8	26.0	32.0	19
15	L.3	9.5	10.0	5	L.4.5	11.5	12.0	4	L.6.5	13.9	16.5	16	L.8	15.0	18.5	19
								(b) Under Covered Portion								
0	L.3	15.5	17.5	11	L.4.5	18.5	19.0	3	L.6.5	22.0	22.2	1	L.8	18.5	25.0	26
2	L.3	19.0	23.0	17	L.4.5	18.5	27.0	31	L.6.5	22.0	29.5	25	L.8	18.5	33.5	45
5	L.3	16.5	20.5	19	L.4.5	18.5	26.5	30	L.6.5	22.0	30.5	28	L.8	18.5	34.0	46
10	L.3	9.5	12.0	21	L.4.5	13.0	16.0	19	L.6.5	15.0	20.5	27	L.8	14.0	22.0	36
15	L.3	4.8	5.5	13	L.4.5	5.5	9.3	41	L.6.5	9.2	11.5	20	L.8	9.2	12.0	23

Comparison of depth dose in r for 100 r in air, at edge of field with that at center of field, 400 kv., half-value layer 3.3 mm. Cu, 60 cm. target-skin distance, 40 per cent transmission grid, for different field areas (*a*) under opening, (*b*) under covered portion.

TABLE III

200 kv.

COMPARISON OF DEPTH DOSE AT EDGE OF FIELD WITH THAT AT CENTER OF FIELD; R FOR 100 R IN AIR, 40 PER CENT TRANSMISSION GRID, T.S.D. 50 CM., H.V.L. 0.94 MM. Cu

Depth in cm.	Area of Field in sq. cm.											
	100				200				400			
	Dis. from cen.	r edge	r cen. beam	% diff. c. beam	Dis. from cen.	r edge	r cen. beam	% diff. c. beam	Dis. from cen.	r edge	r cen. beam	% diff. c. beam
						(a) Under Opening						
0	R.4.5	107	113	5.3	R.6.5	109	116	6	R.9	91	125	27
5	R.4.5	50	55	9	R.6.5	54	60.8	11	R.9	54.5	66.5	18
10	R.4.5	18	23	22	R.6.5	25	27.7	10	R.9	23.5	31.0	24
15	R.4.5	8	10.2	21	R.6.5	10.7	12.5	14	R.9.5	12.5	15.0	17
						(b) Under Covered Portion						
0	R.4.5	16	18.5	13	R.6.5	17.9	23	22	R.9	18.2	25	27
5	R.4.5	16	22.0	27	R.6.5	21.4	27.7	23	R.9	18.2	32.7	44
10	R.4.5	9	12.5	28	R.6.5	14	17.0	18	R.9	18.2	22.0	17
15	R.4.5	4.5	6	25	R.6.5	7.1	8.8	19	R.9.5	8.9	11.8	25

Comparison of depth dose in r for 100 r in air, at edge of field with that at center of field, 200 kv., half-value layer 0.94 mm. Cu, 50 cm. target-skin distance, 40 per cent transmission grid, for different field areas (*a*) under opening, (*b*) under covered portion.

the difference between the edge and the center is largest for the largest field, 400 sq. cm.

Under the open area there is approximately a 5 per cent difference at the surface for a 100 and 200 sq. cm. field, while at a depth of 10 cm. the difference is 20 and 10 per cent respectively. For the 400 sq. cm. field, the difference between the edge and the center is approximately 25 per cent from the surface through a depth of 10 cm.

Under the covered portion on the surface, the difference varies from approximately 15 to 25 per cent from a field size of 100 to 400 sq. cm. The greatest difference is at a depth of 5 cm., varying from 25 to 45 per cent. The difference at a depth of 10 cm. is roughly 20 per cent.

Here, too, because of the fall off at the edge, the field should be large enough to give the periphery of the tumor an adequate dose.

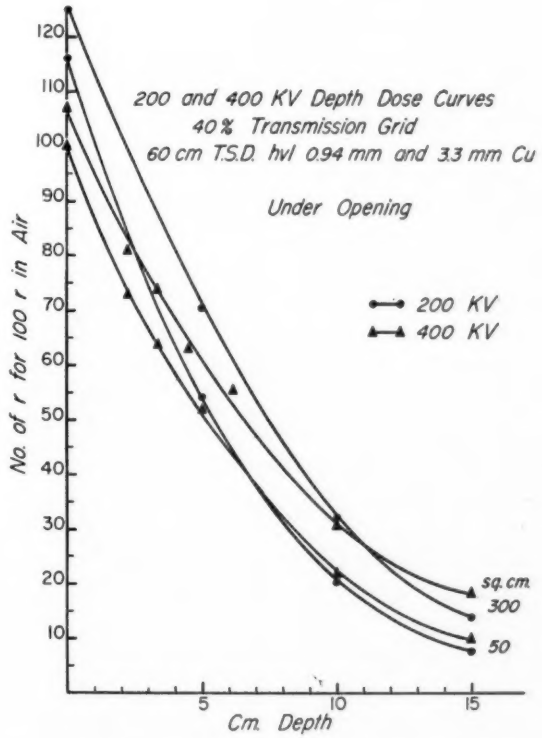

Fig. 5. Curves showing comparison under opening of depth doses between 200 and 400 kv., half-value layer 0.94 mm. Cu and 3.3 mm. Cu, target-skin distance 60 cm., 40 per cent transmission grid.

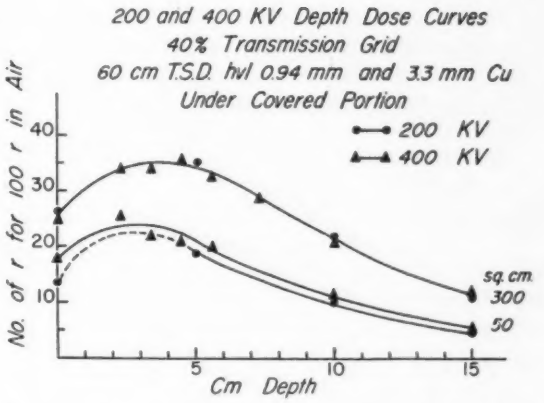

Fig. 6. Curves showing comparison under covered portion of depth doses between 200 and 400 kv., half-value layers 0.94 mm. Cu and 3.3 mm. Cu, target-skin distance 60 cm., 40 per cent transmission grid.

Comparison of 400 and 200 kv.

The depth doses for the central beam under the opening for both 200 and 400 kv. are plotted in Figure 5 for a 50 sq. cm. field and a 300 sq. cm. field. For a 300 sq. cm. field, the depth doses, each expressed as a per cent of the air dose, are larger for the 200 kv. roentgen rays than for the 400 kv. roentgen rays to a depth of about 10 cm., after which the depth dose is greater for 400 kv.

For a field of 50 sq. cm., the depth dose of the 200 kv. roentgen rays is greater to a depth of about 7 cm., after which the depth dose of 400 kv. roentgen rays is greater.

Under the covered area the differences between the 200 and 400 kv. depth doses are very small (see Fig. 6). Only one curve can be drawn through both sets of points for the 300 sq. cm. field. For the 50 sq. cm. field the depth doses of the 400 kv. roentgen rays are only slightly larger than for 200 kv. roentgen rays.

The depth doses, r for 100 r in air, for 400 kv. are compared with those for 200 kv. roentgen rays in Table IV, for the central beam under the opening and under the covered portion, and for the average. The depth dose of the average is greater for 200 kv. radiation than for 400 kv. radiation

TABLE IV

COMPARISON OF 200 AND 400 KV. DEPTH DOSES AT 60 CM. T.S.D., H.V.L. 0.94 MM. Cu AND 3.3 MM. Cu; 40 PER CENT TRANSMISSION GRID, R FOR 100 R IN AIR

Depth in cm.	Area of Field in Square Centimeters							
	50		100		200		300	
	200 kv.	400 kv.	200 kv.	400 kv.	200 kv.	400 kv.	200 kv.	400 kv.
				(*a*) *Under Opening*				
0	116.0	100.0	118.0	104.0	123.5	106.0	124.5	107.0
1	101.5	87.5	105.3	91.5	110.0	94.5	112.0	97.0
2	88.0	76.0	93.5	80.0	98.5	84.0	101.0	87.5
3	75.7	66.0	82.3	70.0	87.5	74.0	90.3	77.5
4	64.0	57.5	71.5	61.0	77.2	64.5	80.0	69.0
5	54.5	50.0	61.7	53.0	67.3	56.0	70.5	60.5
6	46.2	43.0	52.8	46.5	58.3	49.5	61.3	53.5
7	38.8	36.5	44.8	40.0	50.0	44.0	52.5	47.0
8	32.0	31.0	37.5	35.0	42.5	38.5	45.0	41.5
9	26.2	26.0	31.3	30.0	35.8	34.0	38.0	36.0
10	21.0	22.0	25.5	26.0	29.7	30.0	31.7	32.0
11	17.0	19.0	21.0	22.5	25.0	26.8	26.5	28.5
12	13.5	16.6	17.3	19.5	20.8	23.8	22.2	25.5
13	10.8	14.3	14.3	16.8	17.5	21.2	18.8	23.0
14	9.0	12.0	12.2	14.4	15.0	18.8	16.0	20.5
15	7.8	10.0	10.8	12.0	13.3	16.5	14.0	18.5
				(*b*) *Under Covered Portion*				
0	13.5	17.5	21.5	19.0	23.0	22.0	26.0	25.0
1	20.0	21.5	23.8	24.5	26.8	26.5	31.0	30.5
2	22.2	23.0	24.8	27.0	28.8	29.5	33.5	33.5
3	22.5	23.5	25.0	28.0	29.5	30.5	34.8	35.0
4	21.8	22.5	24.3	28.0	29.2	31.0	35.0	35.0
5	20.0	20.5	23.5	26.5	28.2	30.5	34.3	34.0
6	18.3	18.5	21.8	23.5	26.7	29.0	33.0	32.5
7	16.2	16.5	19.7	21.0	25.0	27.0	30.7	29.5
8	14.0	15.0	17.5	19.5	22.8	24.5	28.0	27.0
9	12.2	13.5	15.0	17.5	20.5	22.5	25.0	24.5
10	10.5	12.0	13.0	16.0	18.2	20.5	21.3	22.0
11	9.2	10.5	11.3	14.5	16.0	18.5	18.6	19.7
12	8.0	9.2	9.8	13.2	14.0	16.5	16.3	17.5
13	7.0	8.0	8.7	12.0	12.0	15.0	14.3	15.6
14	5.5	6.6	7.5	10.5	10.3	13.0	12.5	14.0
15	4.5	5.5	6.7	9.5	9.0	11.5	11.0	12.0

Comparison of depth doses in r for 100 r in air, for 200 and 400 kv. at the center of the field, half-value layers 0.94 mm. Cu and 3.3 mm. Cu, 60 mm. target-skin distance, 40 per cent transmission grid, for different field areas (*a*) under opening, (*b*) under covered portion, (*c*) average.

TABLE IV—(*continued*)

Depth in cm.	Area of Field in Square Centimeters							
	50		100		200		300	
	200 kv.	400 kv.	200 kv.	400 kv.	200 kv.	400 kv.	200 kv.	400 kv
	(*c*) *Average*							
0	54.5	50.5	60.3	53.0	63.5	55.7	65.5	57.8
1	51.3	47.9	56.8	51.3	60.5	53.7	63.5	57.1
2	47.3	44.2	52.7	48.2	57.2	51.3	61.0	55.1
3	43.0	40.5	48.0	44.8	53.3	47.9	57.5	52.0
4	38.2	36.5	42.5	41.2	49.0	44.4	53.5	48.6
5	32.7	32.3	37.5	37.1	43.8	40.7	49.0	44.6
6	28.5	28.3	32.3	32.7	38.5	37.2	44.0	40.9
7	24.5	24.5	27.7	28.6	33.8	33.8	39.0	36.5
8	21.0	21.4	23.7	25.7	29.7	30.1	34.3	32.8
9	17.7	18.5	20.2	22.5	26.0	27.1	29.8	29.1
10	14.7	16.0	16.8	20.0	22.7	24.3	25.8	26.0
11	12.0	13.9	14.2	17.7	19.7	21.8	22.2	23.2
12	10.0	12.2	12.0	15.7	17.0	19.3	19.2	20.7
13	8.5	10.5	10.5	13.9	14.2	17.5	16.3	18.6
14	7.3	8.8	9.2	12.1	12.5	15.3	14.0	16.6
15	6.7	7.3	8.7	10.5	10.7	13.5	12.5	14.6

up to a depth of 8 cm., after which the 400 kv. depth dose is larger.

If the surface dose under the openings is considered 100 per cent, the comparison is made in Table v.

DISCUSSION

The per cent of the surface dose is slightly greater for the 200 kv. radiation under the opening up to a depth of 6 cm., after which it is greater for the 400 kv. radiation, except for a small field of 50 sq. cm., where it is greater for 400 kv. after the first 2 cm.

Under the covered portion, the depth dose of the 400 kv. roentgen rays is greater throughout.

The depth dose of the average is greater for 400 kv. radiation throughout, the difference becoming greater as the depth increases.

From this analysis one may conclude that, if one could position the inflexible lead grid as well as the flexible lead rubber grid, the 400 kv. radiation would be advantageous because of its increased depth dose. Because of the smaller differential absorption between bone and muscle with 400 kv. roentgen rays, it is to be preferred wherever the radiation must penetrate bone. Another reason why it is desirable is that the skin has a greater tolerance for 400 kv. radiation.

A disadvantage of the grid used for 400 kv. radiation is that it is not flexible and cannot make good apposition with the surface skin if the area treated has a large curvature.

The reaction under the openings does not seem to be more severe because of the characteristic radiation of lead. Under the covered part the characteristic radiation is absorbed by the aluminum with which the bottom of the filter is lined.

Skin healing starts at the edge of the field under a covered area. One reason for this may be that the dose here is roughly 20 per cent less than that in the central cov-

TABLE V

COMPARISON OF 200 AND 400 KV. DEPTH DOSES AT 60 CM. T.S.D., H.V.L. 0.94 MM. Cu AND 3.3 MM. Cu, 40 PER CENT TRANSMISSION GRID; R FOR 100 R MEASURED ON THE SURFACE UNDER OPENING

Depth in cm.	Area of Field in Square Centimeters							
	50		100		200		300	
	200 kv.	400 kv.	200 kv.	400 kv.	200. kv.	400 kv.	200 kv.	400 kv.
	(a) Under Opening							
0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1	87.5	87.5	89.3	88.0	89.1	89.1	90.0	90.7
2	75.8	76.0	79.2	77.0	79.7	79.2	81.2	81.8
3	65.3	66.0	69.7	67.3	70.8	69.8	72.5	72.5
4	55.2	57.5	60.6	58.7	62.5	60.8	64.3	65.4
5	47.0	50.0	52.3	51.0	54.5	52.8	56.7	56.5
6	39.8	43.0	44.7	44.7	47.2	46.7	49.3	50.0
7	33.5	36.5	38.0	38.4	40.5	41.5	42.2	44.0
8	27.6	31.0	31.8	33.6	34.4	36.3	36.2	38.8
9	22.6	26.0	26.5	28.8	29.0	32.1	30.6	33.6
10	18.1	22.0	21.6	25.0	24.0	28.3	25.5	29.9
15	6.7	10.0	9.2	11.5	10.8	15.6	11.3	17.3
	(b) Under Covered Portion							
0	11.6	17.5	18.2	18.3	18.6	20.8	20.9	23.3
1	17.2	21.5	20.2	23.6	21.7	25.0	24.9	28.5
2	19.1	23.0	21.0	25.9	23.3	27.8	26.9	31.3
3	19.4	23.5	21.2	26.9	23.9	28.8	28.0	32.7
4	18.8	22.5	20.6	26.9	23.6	29.2	28.1	32.7
5	17.2	20.5	19.9	25.5	22.8	28.8	27.6	31.8
6	15.8	18.5	18.5	22.6	21.6	27.3	26.5	30.4
7	14.0	16.5	16.7	20.2	20.2	25.4	24.7	27.5
8	12.1	15.0	14.8	18.7	18.5	23.1	22.5	25.2
9	10.5	13.5	12.7	16.8	16.6	21.2	20.1	22.9
10	9.1	12.0	11.0	15.4	14.7	19.3	17.1	20.5
15	3.9	5.5	5.7	9.1	7.3	10.9	8.8	11.2
	(c) Average							
0	47.0	50.5	51.1	51.0	51.5	52.5	52.7	54.0
1	44.2	47.9	48.2	49.3	49.0	50.7	51.0	53.4
2	40.7	44.2	44.7	46.2	46.3	48.5	49.0	51.5
3	37.1	40.5	40.7	43.1	43.2	45.1	46.1	48.6
4	32.9	36.5	36.0	39.6	39.7	41.8	43.0	45.5
5	28.2	32.3	31.8	35.6	35.5	38.4	39.4	41.7
6	24.6	28.3	27.4	31.4	31.2	35.1	35.3	38.2
7	21.1	24.5	23.5	27.5	27.4	31.9	31.3	34.1
8	18.1	21.4	20.1	24.7	24.1	28.4	27.5	30.6
9	15.3	18.5	17.1	21.6	21.1	25.6	23.9	27.2
10	12.7	16.0	14.2	19.2	18.4	22.9	20.7	24.3
15	5.8	7.3	7.4	10.1	8.7	12.7	10.0	13.6

Comparison of depth doses in r for 100 r measured on the surface under the opening, for 200 and 400 kv. at the center of the field, half-value layers 0.94 mm. Cu and 3.3 mm. Cu, 60 cm. target-skin distance, 40 per cent transmission grid, for different field areas (*a*) under opening, (*b*) under covered portion, (*c*) average.

ered area, and therefore is below tolerance. The blood supply is also better at the edge of the field.

It is still a moot question at what depth the average depth dose should be considered instead of the separate depth doses under the openings and under the covered areas. When a tumor is treated from two opposing portals, it is very unlikely that the radiation from the open area of one portal will be superimposed on the open area radiation from the other portal and likewise for the scattered radiation under a closed portion. However, it is important to know the range of variation which might occur, and therefore the maximum and minimum depth doses.

It would be more economical for the treatment time to be shorter, and it also might perhaps be better to give grid therapy at a shorter target-skin distance than heretofore used for the following reasons:

1. The divergence of the primary beam through the grid openings would be greater at a shorter distance, and perhaps a greater fraction of the grid could be covered and still have sufficient radiation under the openings reaching the tumor at a depth. Or the same fraction could be left open as at present, but because the divergence is greater, the radiation at a depth would be more uniform. The energy which goes through the surface in a limited area would at a given depth go through a larger area than if the target skin distance were larger. Thus, the radiation at a depth would tend to become more uniform while that at the surface would still be separated into more and less heavily irradiated areas, permitting better skin recovery.

2. Quimby, Marinelli and Farrow[3] have found experimentally that backscatter did not change over a range of target-skin distances from 40 to 150 cm. for roentgen radiation with a half-value layer of 1 mm. Cu. It is reasonable to assume that total scatter will obey the same relationship. If this obtains, the contribution of scatter will be independent of the target-skin distance. Because the scatter increases to a depth of 3 to 4 cm., it will tend to compensate for the greater decrease in depth dose at shorter distances due to the inverse square law.

An investigation at shorter distances might prove fruitful.

SUMMARY

1. A method of measuring the depth dose with a grid for the beam of radiation at the center and at the edge of the field for a 400 kv. machine with a light localizer is described.

2. Curves for the depth doses are given for the portion (a) underneath the central opening and (b) underneath the closed portion for a target skin distance of 60 cm. and a half-value layer of 3.3 mm. Cu. The field varied from 50 sq. cm. to 300 sq. cm.

3. A table of depth doses is given for the conditions described in (2) above and for the weighted average.

4. A comparison is made between the depth dose at the center of the field and at the edge of the field.

5. A method is described for measuring the depth dose at the edge of the field for a 200 kv. machine.

6. A comparison is made between the depth doses at the center and at the edge of the field for 200 kv. radiation with a half-value layer of 0.94 mm. Cu.

7. A comparison is made between the depth doses of 400 kv. radiation, half-value layer 3.3 mm. Cu, and 200 kv. radiation, half-value layer 0.94 mm. Cu at 60 cm. target-skin distance:

(a) when 100 r is given in air

(b) when 100 r is given on the surface under the central opening

8. A tentative reason for skin recovery is hypothesized.

9. It is suggested that because of the greater divergence of the beam at shorter distances, grid therapy might be better given at a shorter distance than heretofore used.

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I wish to express my appreciation to Dr. J. R. Freid for his continued interest in this investigation.

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Vol. 69, No. 6

THE EFFECT OF SELECTIVE SHIELDING OF RABBIT INTESTINE DURING TOTAL BODY IRRADIATION*

By RICHARD S. FARR, LIEUTENANT, MC USN† *and* PETER P. H. DE BRUYN, M.D.
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SINCE the work of Regaud, Nogier and Lacassagne[10] in 1912, investigators have been concerned with the possibility that primary damage to the intestinal tract may be a specific cause of death after total body roentgen irradiation. In brief, the observations to support this concept are: (a) histological evidence that the intestinal epithelium is sensitive to damage by roentgen rays;[9] (b) gastrointestinal disturbances are a prominent part of the symptomatology of irradiation sickness;[11] (c) the blood stream of animals becomes infected with microorganisms normally found in the intestinal tract following total body irradiation;[2,8] (d) animals are more easily killed when roentgen rays are applied to the abdomen than to other parts of the body and, conversely, animals become more resistant to roentgen rays when the abdomen is protected than when other parts of the body are shielded.[1] Of the four observations, only the last mentioned lacks specificity regarding the intestinal tract, since externally applied shielding over the abdomen of an animal must result in at least partial shielding of other tissues and organs besides the intestine. It seemed of interest, therefore, to determine what degree of protection could be afforded an animal by specifically shielding the intestinal tract during otherwise total body irradiation. This report is concerned with the results obtained from studies on rabbits with either the small intestine or colon selectively shielded during irradiation with roentgen rays.

EXPERIMENTAL METHODS

To isolate segments of the intestinal tract, a midline abdominal incision about 2 inches long was made under aseptic conditions and the desired segment of intestine delivered from the abdominal cavity. Special care was taken to leave the appendix inside the rabbit, in view of Jacobson's findings,[7] indicating that shielding of this organ afforded protection to the hemopoietic system. All of the small intestine could be delivered with the exception of the first 10 cm. distal to the stomach and the short segment of ileum attached to the appendix. The greater part of the large intestine could also be mobilized, although the distal segment had to remain in the animal because of the attachment to the dorsal body wall. The rabbit was then placed in the prone position on a plexiglass animal board. The board had a transverse opening across the center, the size of which was controlled by converging sliding panels also made of plastic. A plastic box containing sterile isotonic sodium chloride solution was bolted beneath the opening and the animal was placed on the board so that the isolated segment of intestine hung freely in the saline bath. The hole in the animal board was adjusted with the sliding panels to the smallest size which would not impair the blood circulation to the isolated intestine. Rabbits prepared in this manner were used as operated controls because their situation was identical to the shielded experimental animals except for the lead protection. To shield the intestinal segment from the roentgen rays, a box made of 6.4 mm. lead plate was placed around the plastic one and the plastic sliding panels were replaced by lead. The parts of the board which came in contact with the operative field of the intestine were sterilized by immersion in 1:10,000 aqueous solution of

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TABLE I

DATA FROM UNSHIELDED OPERATED CONTROL GROUP AND UNOPERATED CONTROL GROUP FOLLOWING 1,000 R AND 400 R TOTAL BODY IRRADIATION

	No. Irradiated	Survivors after 30 Days	Average Day of Death	Average Initial Weight	Average Per Cent Weight Loss
1,000 r unoperated controls	6	1	11.4	2,101 grams	16.8
1,000 r operated controls	6	0	11.5	2,261 grams	19.1
400 r unoperated controls	5	5	—	2,375 grams	5.8
400 r operated controls	5	5	—	2,416 grams	12.9

zepherin hydrochloride followed by several rinsings with sterile distilled water.

Fifty-two male rabbits were used in these experiments and all animals, whether operated upon or not, were anesthetized with intravenous nembutal. The radiation factors were as follows: 250 kv. (peak), 15 ma., 1.0 mm. Cu and 3.0 mm. bakelite filters, 2.0 mm. Cu half-value layer; distance from target to midline of animal, 35 inches; dose rate, 14 roentgens per minute in air. The dose rate was measured prior to each exposure with a 100 r Victoreen chamber placed in air at the position occupied by the junction of the board and the intestinal box during the actual exposure. To test the effectiveness of the lead shielding, the dose rate was also measured in air inside the plastic intestinal box, with and without the lead protection in place. Without the lead, the readings were within 1 per cent of the values obtained at a comparable position in air, but with the lead shielding the chamber registered only 0.6 per cent of the reading in air.

Animals were irradiated one at a time because the dose rate would be too low if the field of homogeneous dose distribution was enlarged to cover a pair of rabbits. In lieu of irradiating an experimental and a control rabbit simultaneously, a control rabbit was always irradiated immediately following an experimental animal.

Following exposure to roentgen rays, the animals were weighed twice a week and records were kept on mortality. Rabbits dying within the first three days were not included in the study because it has been shown that deaths during the first three days are not dependent on dose at this level.[5]

RESULTS

To learn the effect of the operative procedure upon weight loss and mortality of rabbits after exposure to roentgen rays, a comparison was made between a group of unoperated controls (anesthetized but unoperated) and a group of operated controls (anesthetized with the small intestine in the plastic box). The 6 rabbits in each group were given 1,000 r. A summary of the results of this experiment is presented in Table 1. The operated controls had no survivors and the average time of death was 11.5 days following roentgen irradiation. The unoperated controls had 1 survivor and the average time of death of the other 5 was 11.4 days. The unoperated group lost less weight than the operated animals.

Because all operated animals died, it was decided to repeat the experiment using 400 r instead of 1,000 r. This dose was chosen because it is just below the point where the fatalities would normallly be expected to occur as a result of roentgen irradiation alone.[5] It was reasoned that any contribution by the operative procedure to the lethality of roentgen radiation would be indicated by deaths in the operated control animals given 400 r. None of the 400 r operated controls died although their greatest weight loss averaged 312 grams as contrasted to an average weight loss of 138 grams for the 400 r unoperated controls. A summary of the results of this experiment is included in Table 1.

From these two experiments it was apparent that the operation did not exert a major deleterious influence on the course of events following exposure to roentgen rays,

TABLE II

DATA FROM SHIELDED SMALL INTESTINE GROUP, SHIELDED COLON GROUP, AND UNOPERATED CONTROL GROUP AFTER 1,000 R TOTAL BODY IRRADIATION

	No. Irradiated	Survivors after 30 Days	Average Day of Death	Average Initial Weight	Average Per Cent Weight Loss
1,000 r unoperated controls	10	5	11.0	2,682 grams	13.6
1,000 r small intestine shielded	10	4	11.0	2,607 grams	15.3
1,000 r colon shielded	10	4	11.0	2,647 grams	15.1

as is shown by the similarity of the average day of death in the operated and unoperated controls after 1,000 r and the lack of deaths in the operated controls after 400 r. On the other hand, the operation was not totally innocuous, as is evident from the greater weight loss in the operated control group than in the unoperated controls. Using these data as a baseline, it was then possible to compare a group of unoperated controls with a group of operated animals with the small intestine shielded and another group of operated animals with the large intestine shielded. There were 10 animals in each of the three groups and all received 1,000 r. The results are given in Table II. There was 1 death less among the unoperated controls than in the other two groups, but the average day of death was identical in all three groups. As noted in the previous control experiments, the weight loss of the operated animals was more than in the unoperated controls. Apparently under the conditions of these experiments, shielding of either small or large intestine does not appreciably reduce the lethality of roentgen irradiation.

DISCUSSION

The present experiments indicate that selective shielding of the rabbit intestine during otherwise total body irradiation, at dose levels not exceeding the LD_{100}, does not decrease the lethal effects of roentgen rays as measured by per cent of mortality, weight loss, or the average day of death.

Jacobson *et al.*[6] have recently reported protection from irradiation death by shielding the exteriorized intestine of the mouse. Not only were fewer deaths observed in the shielded mice, but the hemopoietic system was also protected. The divergence in findings may be based on species differences. Another possible explanation is that Jacobson *et al.* shielded a greater portion of the total intestinal tissue. We shielded first the small intestine and then the colon. On the other hand, since in our experiments appreciable amounts of intestinal tissue were shielded, it would be reasonable to expect some degree of protection unless the protection depended upon shielding a major portion of both sections of the intestine at the same time.

Assuming that under some conditions in some animal species a certain degree of protection resulting from selective shielding of intestinal tissue can be demonstrated, the degree of protection is by no means comparable to that demonstrated by Bond *et al.*[1] in rats as a result of applying external lead shielding to large areas of the abdomen. This indicates that the intestine is not the *critical* organ causing the lethal effects of total body irradiation, although there is evidence that the intestine *contributes* to the postirradiation symptomatology[11] and death.[2,8] Damage to other abdominal organs besides the intestine is of equal or greater significance.

Jacobson *et al.* have demonstrated protection to the hemopoietic system and a reduction in mortality in mice by selective shielding of the spleen. This protection appears to be much greater and more specific than that observed following intes-

tine, liver, head, or hind limb shielding.[6] Edelmann[3] has reported that the mortality in mice is reduced if the adrenals are selectively shielded. Chrom[2] has shown that the ability of the liver and spleen to phagocytize bacteria is reduced following irradiation, and he believes that this decreased phagocytic power plays an important role in the postirradiation bacteremia. He was unable to demonstrate the bacteremia when he irradiated the lower abdomen and shielded the hepatic and splenic area. Gershon-Cohen *et al.*[4] employed small, externally applied lead shields over the lung, liver, and lower abdomen. They conclude that their "results would indicate that lead shielding of small areas offers some protection against whole body irradiation without much difference as to the location of the shielded area." These workers did not include the splenic area in their studies.

In view of the accumulated findings just described, it seems likely that the relative sensitivity of animals following irradiation over the abdomen, and the relative resistance of animals when the abdomen is shielded,[1] can best be attributed to a summation of several "organ effects" rather than to one specific organ.

SUMMARY

Rabbits have been exposed to roentgen irradiation while most of the colon or small intestine was surgically exteriorized into a lead chamber.

The results indicate that the selective shielding of the rabbit intestine during otherwise total body irradiation does not decrease the lethal effects of roentgen rays.

It is suggested that the protection conferred on animals when the entire abdomen is shielded during otherwise total body irradiation can best be attributed to a summation of several "organ effects" rather than to one specific organ.

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Thirty-sixth Annual Meeting: 1954, to be announced.

EDITORIALS

THE ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE Thirty-fifth Annual Meeting of the American Radium Society was held in St. Louis, Missouri on April 20, 21 and 22, 1953, at the Chase and Park Plaza Hotels. The meeting was considered an outstanding success from both the scientific and social aspects. The total attendance was 228, consisting of 81 members, 125 guests and 22 ladies. Following the Address of Welcome by Dr. Robert A. Moore, Dean of the Washington University Medical School and the Presidential Address by Howard B. Hunt, the morning session of the first day was devoted to a group of papers dealing with cancer of the head and neck.

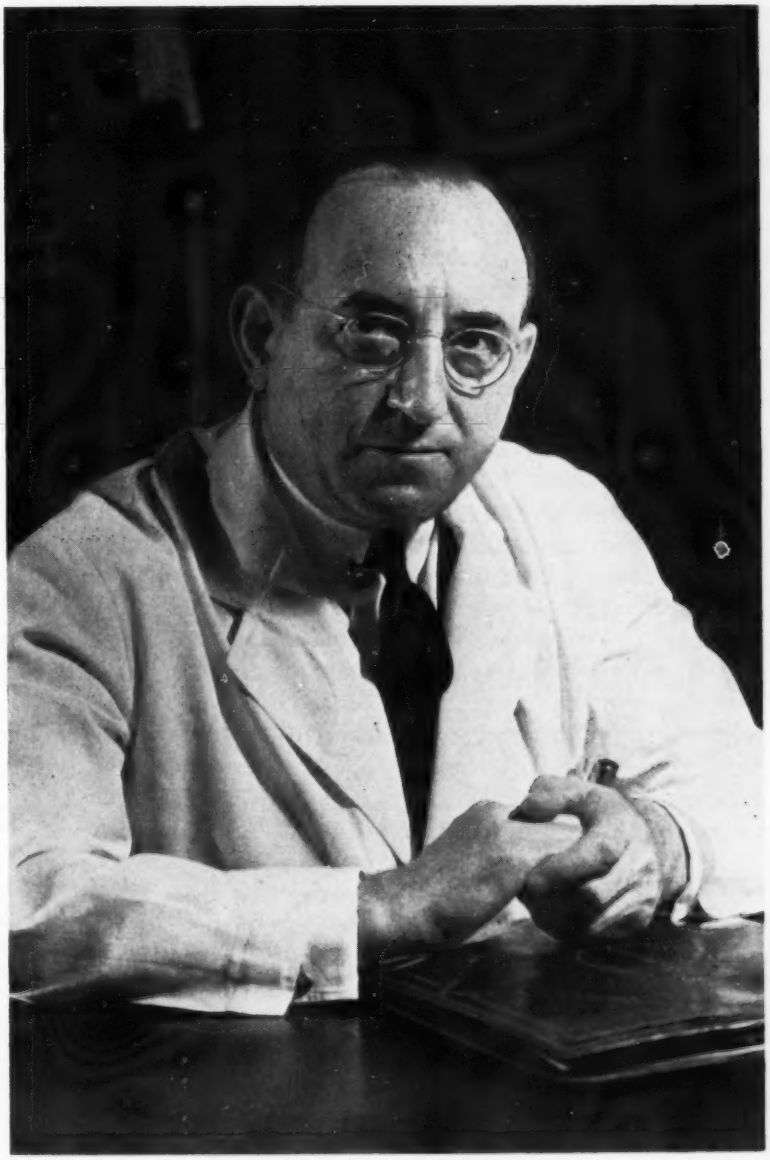
One of the highlights of the meeting was the Janeway Luncheon and Lecture held in the Tiara Room of the Park Plaza Hotel. The Janeway Lecture, an annual event, was given by Dr. Leon O. Jacobson of Chicago on "Factors Concerned in Recovery from Radiation Injury." He was presented the Janeway Medal by Dr. Frederick W. O'Brien in recognition of his outstanding research in the field of radiology.

The scientific program was continued for the next two days. A variety of subjects was covered including various phases of cancer therapy and physics. There was an innovation on the morning of the third day consisting in the presentation of movie and technical demonstrations and by the holding of a tumor conference with panel discussion. The papers presented at the meeting will be published in future issues of this JOURNAL.

The members of the American Radium Society of St. Louis were hosts on Monday, April 20, at a Cocktail Party held in the Tiara Lounge of the Park Plaza Hotel from 6:00 to 7:00 P.M. The Annual Banquet was held on Tuesday evening on the Starlight Roof of the Chase Hotel. Dr. Folke Jacobson of the Radiumhemmet of Stockholm, Sweden expressed his appreciation for being invited as guest speaker at the scientific sessions and for the cordiality shown him by the members. Dr. James T. Case, the only living charter member of the American Radium Society, spoke entertainingly on "Some Early Experiences in Therapeutic Radiology." He related many experiences during the first nine annual meetings of the Society and told interesting anecdotes regarding the crude equipment he used in the early days.

At the close of the meeting on Wednesday noon, the Society was most pleasantly entertained by Dr. and Mrs. Edwin C. Ernst at a noon barbecue at the Lazy M.D. Bar Ranch.

HARRY HAUSER, M.D.



WILLIAM HARRIS
1894-1953

DR. WILLIAM HARRIS, pioneer radiotherapist, chief of the Department of Radiotherapy and chief of the Tumor Clinic of the Mount Sinai Hospital in New York City and also the Morrisania City Hospital, Clinical Professor of Radiology at Columbia University College of Physicians and Surgeons, died on April 7, 1953, at the age of fifty-eight years, after a long illness. His passing is mourned by his many friends and colleagues who knew him also as a grand fellow with a zest for life and a love for all that is fine, beautiful and noble. With his passing we have lost a great leader

in the field of therapeutic radiology, one who has literally grown up with this young specialty and has experienced its maturity into a major field of medicine.

William Harris was born in New London, Connecticut, on October 19, 1894. He was the eldest of six brothers, all of whom have distinguished themselves in various professional fields. He attended Brown University and Harvard Medical School. After receiving his degree of Doctor of Medicine and following his internship at Mount Sinai Hospital, New York, he planned to specialize in gynecology. However, he was persuaded to study roentgen and radium therapy which, in the early twenties, was a relatively new and unexplored field. In order to learn what was then known about radiation therapy, Dr. Harris travelled extensively both in this country and abroad and he visited practically every known radiation therapy center in the world. With knowledge acquired by his own observation and with vision fostered by a pioneer spirit, he developed the Department of Radiotherapy in Mount Sinai Hospital into a major treatment center for cancer, well known and recognized the world over. His interest in cancer led him further to establish the Tumor Clinic in the out-patient department of the hospital. It was his desire to coordinate all our knowledge, our thinking, and our management of cancer for the ultimate purpose of giving better and greater service to the community.

In 1930, while on a visit to the Curie Institute in Paris, Dr. Harris observed the work of Henri Coutard and intuitively recognized the genius of this pioneer in radiotherapy. In the following year, in spite of the pessimism then prevailing, he treated his first case of cancer of the larynx according to the principles of Coutard and the result was a complete success. The success of this first case was the stimulus for a long and extensive period of clinical research in the roentgen treatment of cancer of the larynx, an experience which culminated in the establishment of this form of treatment as the method of choice for this dreaded disease.

William Harris' interests in radiotherapy covered every phase of medicine and surgery and were reflected in the number and variety of original articles published from his department. He established a policy of cooperation with every service in the hospital. The functions of the Department of Radiotherapy were integrated with every other department and specialty. It was the direct result of his concern for the complete welfare of the patient that the Department of Radiotherapy became a great treatment center for cancer patients, not just a clinic for radiotherapy but a center where the best interests of the patient are studied, where surgery, hormone therapy, and chemotherapy hold equal place with roentgen and radium therapy, a center where the social services play an active role in the management of every problem, a center where the guiding spirit is what is best for the individual patient. It is no surprise that the Department of Radiotherapy of Mount Sinai Hospital enjoys a worldwide reputation, that the fame of William Harris has spread everywhere, that visitors come from radiation centers all over the world, and that students arrive from every state and country. Such are the well deserved rewards of a man whose noble outlook on life inspired the high standards of the institution he developed.

Dr. Harris trained many students who passed the examinations and were certified by the American Board of Radiology. He himself belonged to the first group of diplomates of that Board and he was one of the original fellows of the American College of Radiology. He was also a member of the American Roentgen Ray Society, the American Radium Society, the Radiological Society of North America, and the New York Roentgen Society. He was a fellow of the New York Academy of Medicine. He was one of the founders of the New York Cancer Society and was that Society's first treasurer. He was a popular lecturer and his lectures were always enlivened by a fine sense of humor, an inexhaustible collection of good stories, a sound knowledge of the literature, and best of all,

by a severe criticism of his own work.

It was an inspiration to work with William Harris. He allowed and encouraged complete freedom of thought and he was always ready and eager to investigate and develop new ideas. He repeatedly stressed the importance of the individual and the constant need to subordinate any method of treatment, whether radiological or surgical or otherwise, to the over-all picture of the patient himself, his temperament, his capabilities, and his expectation of life. We were constantly reminded not of the life of the patient but of his way of living. Perhaps this stress on the way of living was but a reflection of his own principles of life. He was fond of good, gracious living. He loved good music, good food, and good stories. He was himself a capable musician, a connoisseur of rare foods, and a raconteur with a keen sense of humor.

William Harris is gone but his spirit lives on. All those privileged to have come in contact with him learned the importance of living, the spirit of graciousness and of beauty of life, and the sense of the higher values. He was a man of the world and he gave freely of his worldly wisdom to the many who brought their problems to him. Although he was a specialist in one of the most highly specialized branches of medicine, his opinions on general matters were eagerly sought after by many of his medical colleagues in different branches of medicine. His sound philosophy of life was like a rock of strength and salvation to his family and to his many friends who came to him for advice and guidance in their problems of life. William Harris was recognized by all as a man of deep understanding, of great liberality, and of profound respect for mankind.

Sidney M. Silverstone, M.D.

EARL HAROLD KING
1876-1952

EARL HAROLD KING died on October 22, 1952, from arteriosclerotic cardiovascular disease with hypertension.

Born in Saratoga Springs, New York, to Almeron King and Laura Hodges King on March 16, 1876, he was educated in the public school system of this community. He studied medicine at Eclectic Medical College of New York from which he graduated in 1898. The announcement of Röntgen's epochal discovery made a deep impression upon this young medical student.

He came back to Saratoga Springs in 1898 to enter general practice. In 1902,

however, he returned to New York City for postgraduate study of roentgenology at New York Post-Graduate Medical School and Hospital under Dr. Fred Albee.

In 1909 the roentgen-ray machine at the Saratoga Hospital had completely broken down. Dr. King applied for membership on the medical staff and offered to equip and operate a modern roentgen department. Not until 1911 was formal approval given to this offer. At that time plans were being made for a new hospital building which opened in 1913. From then until his retirement on December 1, 1949, Dr. King provided uninterrupted diagnostic and therapeutic roentgenologic service, supplying and maintaining all equipment and accessories, technical and clerical service at his own expense which was often discouragingly large in relation to the income derived. Nonetheless he gave freely of his time and materials to charity cases. Finally when the depression of the 1930s was at its worst, the greatly increased volume of charity work forced him to make a charge adequate to cover cost of materials only. Saratoga Hospital was extremely fortunate to have on its staff a true pioneer disciple of Röntgen and a physician whose zeal to provide his community and his hospital with the best therapeutic and diagnostic facilities transcended the normal urge to accumulate money. His uncompromising professional standards were largely responsible for preserving to this day strictly ethical and approved practice in conduct of the Department of Radiology despite several attempts by uncomprehending lay boards of managers to substitute the familiar employee relationship.

Besides contributing so much to the development of Saratoga Hospital, Dr. King was active in civic and fraternal affairs. He was long term trustee of the First Baptist Church. He served fifteen years on the Board of Education and for a like period on the Board of Directors of the Y.M.C.A. He was a 32nd Degree Mason and former Commander of Washington Commandery, Knights Templar, as well as a Past Exalted Ruler of Elks Lodge No. 161.

Professionally he served for nine years as Examiner in Pathology on the New York State Medical Licensing Board. He was a diplomate of the American Board of Radiology, fellow of the American College of Radiology, member of the American Roentgen Ray Society, the Radiological Society of North America, and the Eastern Urological Society of the State of New York, the American Medical Association, the New York State Medical Society, the Saratoga County and Saratoga Springs Medical Societies. Over the years he occupied numerous offices, including the presidency, of these last two societies where his regular attendance, unflagging interest, minute knowledge of parliamentary procedure, and love of a good story served unfailingly to stimulate and to enlighten. He was a born raconteur. It was a rare occasion indeed when Dr. King had no tale to relate.

With his passing, medicine in general and radiology in particular have lost another stout advocate of free enterprise in medicine.

Robert S. Hayden, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 29–Oct. 2, 1953.

AMERICAN RADIUM SOCIETY
Secretary, Dr. R. E. Fricke, Mayo Clinic, Rochester, Minn. Annual meeting: 1954, to be announced.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill., Dec. 13–18, 1953.

AMERICAN COLLEGE OF RADIOLOGY
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: New York, New York, June 1–5, 1953.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. R. Lee Foster, 15 E. Monroe, Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Rayle, Jr., 478 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, October through April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. F. A. Riebel, 15 West Goodale St., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Columbus Athletic Club, Columbus, Ohio.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Elbert K. Lewis, 6337 S. Harvard Ave., Chicago, Ill. Meets second Thursday of each month, October to April inclusive at the University Club.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Mortimer Lubert, 10515 Carnegie Ave., Cleveland 6, Ohio. Meetings at 6:45 P.M. on fourth Monday of each month from October to April.

COLORADO RADIOLOGICAL SOCIETY
President, Dr. W. P. Stampfli, St. Luke's Hospital, Denver, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday October and April.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. J. C. Cook, Harper Hospital, Detroit, Mich. Meets monthly first Thursday, October to May, at Wayne County Medical Society Building.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. A. Judson Graves, 2002 Park St., Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. Robert M. Tankesley, 218 Doctors Bldg., Atlanta, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary, Dr. E. H. Bryson, 273 Alhambra Circle, Coral Gables, Fla. Meets third Wednesday of each month at Veterans Administration Building.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Harry Fishbein, 324 Medical Arts Bldg., Houston 2, Texas. Meets fourth Monday each month at Hermann Hospital.

IDAHO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred M. Stone, St. Lukes Hospital, Boise, Idaho. Meets annually at same time as Idaho State Medical Association.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

IOWA X-RAY CLUB
Secretary, Dr. James McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Willis L. Beeler, M.D., 700 Kansas Ave., Topeka, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430 48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif. Meets second Wednesday each month at Los Angeles County Medical Association Building, Los Angeles.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Maine Radiological Society
Secretary, Dr. Hugh Allan Smith, Eastern Maine General Hospital, Bangor, Maine. Meets in early spring, June and fall.

Miami Valley Radiological Society
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. T. J. Pfeffer, 839 N. Marshall St., Milwaukee 2, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. John R. Hodgson, Mayo Clinic, Rochester, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

Mississippi Radiological Society
Secretary, Dr. John W. Evans, 117 N. President St., Jackson, Miss. Meets third Thursday of each month at the Rotisserie Restaurant at 6:30 P.M.

Montana Radiological Society
Secretary, Dr. Grant P. Raitt, Suite 413 Medical Arts Bldg., Billings, Montana. Will meet at least once a year.

Nassau Radiological Society
Secretary, Dr. Joseph J. La Vine, 259 North Grand Ave., Baldwin, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Stanley M. Wyman, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Harvard Club.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. J. R. Freid, 1049 Park Ave., New York 28, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings by announcement.

Northeastern New York Radiological Society
Secretary, Dr. John F. Roach, Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesdays of October, November, March and April. Annual meeting will be held in May or June, 1953.

Northern California Radiological Society
Secretary, Dr. Richard C. Ripple, 1215 28th Street, Sacramento, Calif. Meets at dinner last Monday of September, November, January, March and May.

Ohio State Radiological Society
Secretary, Dr. Willis S. Peck, 1838 Parkwood Ave., Toledo 2, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. John R. Danstrom, Medical Arts Bldg, Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. J. Richard Raines, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse 416 Pine St., Williamsport. Annual meeting, Necho Allen Hotel, Pottsville, Pa., May 22 and 23, 1953.

Philadelphia Roentgen Ray Society
Secretary, Dr. George P. Keefer, American Oncologic Hospital, Philadelphia 4, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Donald H. Rice, Western Pennsylvania Hospital, Pittsburgh 24, Pa. Meets 6:00 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Radiological Section, Baltimore Medical Society
Secretary, Dr. H. Leonard Warres, 2337 Eutaw Place, Baltimore 17, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Section, Connecticut Medical Society
Secretary, Dr. William A. Goodrich, 85 Jefferson St., Hartford 6, Conn. Meetings are held bi-monthly.

Radiological Section, Medical Society of the District of Columbia
Secretary, Dr. U. V. Wilcox, 915 Nineteenth St., N. W., Washington 6, D. C. Meets Medical Society Library, third Thursday of January, March, May and October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr Roy G. Giles, Temple, Texas.

Radiological Society of Greater Cincinnati
Secretary, Dr. Harry K. Hines, 2508 Auburn Ave., Cincinnati 19, Ohio. Meets monthly from September to June on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Colonel Alexander O. Haff, M.C., Tripler Army Hospital, Honolulu, T. H. Meets third Friday of each month at Tripler Army Hospital.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of New Jersey
Secretary, Dr. Nicholas G. Demy, 912 Park Avenue, Plainfield, N. J. Meets annually at Atlantic City at time of State Medical Society and mid-winter at Elizabeth, N. J.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. A. Gordon Ide, 277 Alexander St., Rochester 7, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Next annual meeting, Shirley-Savoy Hotel, Denver, Colo., August 20, 21 and 22, 1953.

St. Louis Society of Radiologists
Secretary, Dr. Francis O. Trotter, Jr., 634 N. Grand Blvd., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August and September.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets Brook Army Medical Center Officers Mess first Monday of each month.

San Diego Radiological Society
Secretary, Dr. C. W. Bruner, 2456 Fourth Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. I. J. Miller, 2680 Ocean Avenue, San Francisco 27, Calif. Meets quarterly.

Section on Radiology. California Medical Association
Secretary, Dr. Calvin L. Stewart, 2330 First Ave., San Diego 1, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. George E. Irwin, Jr., 703 N. East St., Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Bay Radiological Society
Secretary, Dr. W. H. Graham, 634 East Santa Clara St., San Jose 12, Calif. Meets second Wednesday each month.

South Carolina Radiological Society
Secretary, Dr. Henry E. Plenge, Spartanburg General Hospital, Spartanburg, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, Newell Hospital, Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort worth 4, Texas. Next meeting, Dallas, Texas, Jan. 29 and 30, 1954.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. John N. Burkey, 555 Medical-Dental Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May.

Westchester Radiological Society
Secretary, Dr. Clifford C. Baker, Harwood Bldg., Scarsdale, N. Y. Meets on third Tuesdays of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Irving I. Cowan, 425 E. Wisconsin Ave., Milwaukee 2, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. Wm. W. Saunders, Veterans Administration Hospital, San Francisco 21, Calif. Meets monthly, third Thursday, at 7:45 P.M., Lane Hall, Stanford University Hospital.

Cuba, Mexico and Central America

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86, Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. D. L. McRae, Suite 305, 1555 Summerhill Avenue, Montreal 25, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

College of Radiologists (Australia and New Zealand)
Honorary Secretary, Dr. Alan R. Colwell, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W.

South America

Asociacion Argentina de Radiologia
Secretary, Dr. Lidio G. Mocsa, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Sociedad Argentina de Radiologia, Junta Central, Buenos Aires
Secretary, Dr. Oscar F. Noguera, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Argentina de Radiologia, Filial del Litoral, Sede, Rosario
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 19 ha 663 Italia St., Rosario, Santa Fe.

Sociedad Argentina de Radiologia, Filial del Sur, Sede, La Plata
President, Prof. Dr. Jose P. Uslenghi, La Plata. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Centro Sede, Cordoba
President, Prof. Dr. David L. Caro, Corro 86, Cordoba. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Norte, Sede, Tucuman
President, Dr. Julio Mendez Valladares, Rivadavia 358, Tucuman. Meets monthly.

Sociedad Argentina de Radiologia, Filial de Cuyo Sede, Mendoza.
President, Dr. J. A. Orfila, Hospital Central, Mendoza. Meets monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Colombiana de Radiologia
Secretary, Dr. Luis E. Lozano B., Carrera 12 No. 20–69, 6º piso, Bogota, Colombia. Meets last Thursday of each month.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Allies, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Société Française d'Electro-Radiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord
Secretary-General, Dr. A. Dariaux, 9 bis, Boulevard Rochechouart, Paris IX^{e}, France.

Ceskoslovensk společnost pro rëntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10:30, X-Ray Dept., Akademia Gdansk.

Wrocław Section Polish Society of Radiology
Secretary, Dr. R. Marciniak, Akad. Medyczna Wrocław, 68 Curie-Skłodowska St., Wrocław, Poland. Meets first Sunday of each month, X-Ray Department, Akademia Medyczna Wrocław.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Deutsche Roentgen-Gesellschaft
Secretary, Dr. Peter Kröker, (22a) Essen-Helsingen, Westpreussenstrasse 21. Meets annually in different towns.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

FOURTH INTER-AMERICAN CONGRESS OF RADIOLOGY

The Fourth Inter-American Congress of Radiology was held in Mexico City November 2 to 8, 1952. Prior meetings of the Inter-American Congress of Radiology were held in Buenos Aires (1943), Habana (1946), and Santiago, Chile (1949). From the United States radiologists and their families attended the Mexican meeting to the number of 287, and we understand that over 600 radiologists and members of their families from the New World were registered.

In addition to congressists from the New World there were present a few invited guests from Germany, Belgium, Holland, England, Sweden, and Switzerland. Most of the German and Dutch registrants were connected with the large German and Dutch roentgen-ray commercial exhibits. The United States official delegation consisted of eight appointees of the various radiological organizations and eight from the Federal Government Agencies. The delegates included: Paul C. Aebersold (Atomic Energy Commission); Joseph C. Bell; Colonel G. C. Bess (Department of Defense); Howard P. Doub; Gioacchino Failla (Atomic Energy Commission); Lowell S. Goin; Ross Golden; Philip J. Hodes; Maurice Lenz; Egon Lorenz (National Cancer Institute); Eugene P. Pendergrass; Edith H. Quimby (American Radium Society and the Atomic Energy Commission); Juan A. del Regato; Shields Warren (Atomic Energy Commission), and James T. Case, Chairman of the Official Delegation.

The meetings were held under the Presidency of our esteemed mutual friend, Dr. Manuel F. Madrazo. The Vice President was Dr. Jose Araugo G., President of the Mexican Society of Radiology. All the members of this Society participated actively in the arrangements, having been assigned to the various committees. The Ladies' Committee was especially distinguished for its efficiency and industry. Their efforts not only provided special entertainment but also sight-seeing visits to a number of scenic and historic localities.

Dr. Lowell S. Goin had been commissioned by the American College of Radiology to arrange a special convocation to present a certificate of honor to Dr. Manuel F. Madrazo, President of the Congress, who for years had been an honorary member of the American College of Radiology but had never passed through the formal convocation. This was a notable event, and long to be remembered.

The scientific program was carried out very successfully, thanks to International Business Machines who loaned its electronic equipment for simultaneous interpretation and a corps of exceptionally competent interpreters. A large part of the proceedings of the Congress took place under these favorable circumstances. There were very instructive scientific exhibits and some fine commercial exhibits in which both American and European firms participated.

The official themes of the Congress related to the diagnosis of intra-abdominal tumors other than those involving the digestive tube, and the treatment of lymphoblastomas. These were discussed by a number of delegates and final conclusions were published at the close of the meeting. The other contributions to the Congress were listed as "free papers." Special interest attached to the discussion of radioisotopes, and particularly the employment of cobalt 60. Much time was given to the discussion of radiotherapy of cancer. Numerous contributions on roentgen diagnosis were presented, too numerous to be mentioned here. The Organizing Committee proposes to publish the Proceedings of the Congress in *La Revista Mexicana de Radiologia* ($5.00 per year, Medellin No. 86, Mexico 7, D.F.). This is a very much worthwhile journal, with most of its illustrations in commendable half tones, reproduced as actual photographs of reduced size.

In the final assembly, Washington, D. C., was the choice for the meeting of the Fifth Inter-American Congress of Radiology, to take place during the week of April 24, 1955. The officers elected for the next Congress include: James T. Case, President; Lowell S. Goin, Executive Vice President; Eugene P. Pendergrass, General Secretary; Philip J. Hodes, Secretary for the United States; Juan A. del Regato, Secretary for Latin America; and Earl E. Barth, Treasurer. This group constitutes the nucleus of the organizing committee for the Fifth Inter-American Congress of Radiology in Washington, D. C., in 1955. The Shoreham Hotel has been secured for the meeting. It is anticipated that this will be an outstanding meeting and that there will be a large attendance from Latin America as well as from the United States and Canada.

This Congress was organized by and runs

under the auspices of the *Inter-American College of Radiology* which is now a well established and going concern, including all the organized radiologists in Latin America. There are also numerous participants from the United States and Canada. The President of the College is Dr. Pedro A. Maissa, whose address is the same as the Executive Secretary (1516 Tucuman, Buenos Aires, Argentina). The College publishes a journal called "*Acta Radiologica Inter-Americana*," which is distributed to the membership. Initiation fee, five dollars; annual dues, five dollars. As interest in Latin America increases among us, it is hoped that many radiologists from the United States and Canada will feel impelled to join the Inter-American College of Radiology. Application blanks may be secured from Dr. Juan A. del Regato, Inter-American College Counsellor for the United States, Penrose Cancer Hospital, Colorado Springs, Colorado.

James T. Case, M.D.

OAK RIDGE SUMMER SYMPOSIUM

The Fifth Annual Oak Ridge Summer Symposium will be devoted to modern physics this year after a three year concern with other fields of science. The symposium will be held from August 24 to 29, 1953, and will consist of several lectures within the general fields of mesons and ultra high energy phenomena, on the one hand, and electromagnetic radiations, on the other.

Speakers on the symposium will include: Jesse W. Du Mond, California Institute of Technology; Ralph Livingston, J. Rand McNally, and T. A. Welton of the Oak Ridge National Laboratory; M. Stanley Livingston, Massachsetts Institute of Technology, and J. Steinberger, Columbia University. Specific subjects to be covered include mesons, cosmic rays, micro-wave, infra-red and optical spectra, gamma-ray spectroscopy, quadrupole moments, and quantum electrodynamics.

The formal lecture sessions will be held in a theater building in downtown Oak Ridge and will run from 8:30 A.M. to 12:30 P.M. daily. Several other activities are being planned in connection with the symposium, including a tour of unclassified physical research facilities at Oak Ridge National Laboratory, a banquet, and an outing at Big Ridge on Norris Lake.

This will be the second in the series of Oak Ridge summer symposiums to be devoted to modern physics. The first symposium five years ago was concerned with this field, and subsequent gatherings have been devoted to a review of modern chemistry and discussions of the role of atomic energy in engineering and in agriculture.

The symposiums are sponsored jointly by the Oak Ridge National Laboratory and the Oak Ridge Institute of Nuclear Studies. Additional information may be obtained from the University Relations Division of the Institute, P.O. Box 117, Oak Ridge, Tennessee.

REGISTRY OF RADIOLOGIC PATHOLOGY AT THE ARMED FORCES INSTITUTE OF PATHOLOGY

This is to remind radiologists of the services that are being rendered by the Registry of Radiologic Pathology, Washing, D. C., sponsored by the American Roentgen Ray Society, the Radiological Society of North America, and the American College of Radiology.

As is known, the Registry is the national archives for interesting cases of teaching value to which roentgenologists throughout the country are willing to contribute for its growth.

Participation in this growth is earnestly solicited by forwarding to the Registry cases of this type. It is only in this way that a continuous supply of material can be accumulated for study, classification, analysis and correlation with pathologic findings. The information gained from this processing is reflected back to the medical profession in the form of loan sets, exhibits, and research facilities.

Collections, prepared exhibits, and reported cases are most welcome. In addition to the roentgenograms, the Registry requests a brief clinical abstract of the case and the histopathology slides which help to establish the diagnosis. If possible, colored photographs of the gross pathology are also very desirable.

The original roentgenograms will be duplicated on 8 by 10 inch transparencies to be filed in the Registry. The contributor's roentgenograms will be returned to him. The slides of the microsection will be kept in order to supplement the future

usefulness of the case. (If it is more convenient to send the tissue blocks, the Armed Forces Institute of Pathology will make their sections, after which the blocks will be returned.)

The material should be addressed to: The Director, Armed Forces Institute of Pathology, Attention: Radiologic Registry, Washington 25, D. C.

If further information is desired concerning the activities or services offered by the Radiologic Registry, address: The Registrar, Radiologic Registry, Armed Forces Institute of Pathology, Washington 25, D. C.

Inter-Society Committee for the Registry of Radiologic Pathology
John D. Camp, M.D., *Chairman*
Paul C. Swenson, M.D.
Aubrey O. Hampton, M.D., *Secretary*

NEW OFFICERS AMERICAN RADIUM SOCIETY

At the Thirty-fifth Annual Meeting of the American Radium Society held in St. Louis on April 20, 21 and 22, 1953, the following officers were elected for the coming year: *President-Elect:* Dr. John E. Wirth, Pasadena, California; *1st Vice-President:* Dr. H. Dabney Kerr, Iowa City, Iowa; *2nd Vice-President:* Dr. Grant H. Beckstrand, Long Beach, California; *Secretary:* Dr. Robert E. Fricke, Rochester, Minnesota; *Treasurer:* Dr. Douglas J. Roberts, Hartford, Connecticut. The *President* is Dr. Edith H. Quimby, New York, New York.

HOUSTON RADIOLOGICAL SOCIETY

At the January, 1953, meeting of the Houston Radiological Society, the following officers were elected for the coming year: *President,* Dr. Thomas G. Russell; *Vice-President,* Dr. Frank M. Windrow; *Secretary,* Dr. Harry Fishbein, 324 Medical Arts Bldg., Houston 2, Texas; *Treasurer,* Dr. D. M. Earl.

Vol. 69, No. 6

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Marañón, G., Martínez Díaz, J., and Muñoz Larrabide, J. Un case de síndrome de Crouzon. (A case of Crouzon's syndrome.) *Bol. Inst. Pat. Méd.*, May, 1952, *7*, 81–84.

In 1912, O. Crouzon, from the Salpetrière, published a case of "hereditary craniofacial dysostosis." This case was characterized by oxycephaly, exophthalmos, strabismus, diminished visual acuity and prognathism. In his book on familial disease he also described some cases unrelated to heredity.

Cases like the present are rare, and their clinical signs are referable to ocular manifestations due to bony alterations. The case is that of a six year old girl without a remarkable family history. At birth she was a blue baby. Her exophthalmos developed very rapidly and the visual acuity diminished accordingly. Roentgenograms of the skull showed the typical appearance described by Crouzon. The sutures showed increased density and were sunk in relation to the bulging of the different bones that they limit. The brain markings of the inner table of the skull were very prominent. There was also platybasia, enlargement of the sella, and thinning of its dorsum. The facial bones appeared pushed back in relation to the frontal bone. The maxillary bone and jaw were underdeveloped, accounting for the irregularity of the dental alignment.

The reported case was not related to heredity and it is an error to consider every instance as such. Crouzon's syndrome is an embryopathy that can appear sporadically due to alterations of the embryo or can be transmitted by heredity to others. The diagnosis, which is usually made by roentgen examination, is simple, although the condition can be mistaken for leontiasis or multiple exostosis.

The relation to endocrinopathy was demonstrated in the case described for the first time by Crouzon since the mother had goiter. In the present case the alterations of the sella turcica were interpreted as due to the pituitary hypothalamic change.—*Julio Toriello, M.D.*

Dewulf, L. Deux cas d'épithélioma épibulbaire guéris par la radiothérapie de contact. (Two cases of epibulbar epithelioma cured by contact roentgen therapy.) *J. belge de radiol.*, 1952, *35*, 107–112.

Epibulbar epithelioma is a rare tumor usually localized to the corneoscleral junction of the eye. It is pearly white and firm and tends to spread over the surface of the bulb but it does not infiltrate deeply. Conservative surgical procedures are unsatisfactory because of frequent recurrences, while radical surgery requires loss of the eye by enucleation. Ordinary roentgen therapeutic methods are not suitable because uninvolved tissues cannot be spared. Radium therapy cannot be applied well because of the length of time required to maintain the eye motionless at each treatment, and with radium there is also the risk of late complications. The necessary physical requirements for the roentgen treatment of epibulbar epithelioma are high intensity and low penetration. These requirements are met by the contact roentgen apparatus.

The author employed a Van der Plaats-Philips machine. With the patient's head immobilized in a head rest, the eye anesthetized, and the lids retracted, immobility of the eye is obtained by fixation of gaze at an appropriately placed object. The treatment field is outlined either by a localizer attachment or a lead cut-out. The Philips tube is easily applied manually. The physical factors are: 50 kv., no filter, target-skin distance of 40 mm. The dose is 5,000 to 6,000 r administered in fractions of 500 r daily or an alternate days. Since the treatment is a matter of seconds, there is no difficulty in maintaining the eye immobile.

Two cases are described by the author. Both responded well to treatment by complete disappearance of the tumor after a mild radiation conjunctivitis. A normal appearance was restored to the eye and there was no interference with vision. During the short follow-up period there were no complications. The danger of opacification of the lens is minimized because of the rapid drop in depth dose with this technique. The author found 9 similar case reports in the literature, some of them treated with the Chaoul apparatus.—*Sidney M. Silverstone, M.D.*

Sears, Victor H. Mandibular condyle migrations as influenced by tooth occlusions. *J. Am. Dent. A.*, Aug., 1952, *45*, 179–192.

The author believes that there is little appreciation of the amount of suffering unnecessarily endured by artificial denture patients because of malrelated condyles. By means of an ingenious device, the condyle migration recorder, he can demonstrate and actually measure the movements of the condyles in all three dimensions. By taking serial readings, eccentric condylar and mandibular relations can be defined and the proper position for centric relations can be determined. Dentures can then be accurately adjusted to bring the mandible and condyles into equilibrium for ideal function.

It is necessary to so establish the occlusion that malrelations will not develop again. The author's method of meeting the problem is to make the first molars of a material more resistant to wear than is used to make the teeth anterior to the first molars. Then if the load tends to migrate forward, the wearing of the anterior teeth will maintain the maximum force on the less readily abraded first molars. Occlusion is thus automatically adjusted over long periods of time.—*George Cooper, Jr., M.D.*

Finochietto, Ricardo, and Yoel, José. Parotiditis y submaxilitis reflejas. (Reflex parotitis and submaxillitis.) *Prensa méd. argent.*, Aug. 8, 1952, *39*, 1846–1849.

The classical theory of the pathogenesis of infection of the salivary glands and ducts is that the ducts are invaded by pathogenic organisms ascending from the mouth. The infection is favored where stasis of the ducts is caused by mechanical obstruction. The inflammatory process causes a desquamation of the epithelium of the acini and ducts. When this material is combined with the minerals in the saliva they form salivary calculi. Subsequently the obstruction causes dilatation and inflammatory changes in the ducts.

Frequently sialograms made of patients without a history of a salivary calculus, but suffering with parotitis and submaxillitis, disclose a dilatation of the salivary ducts. These patients complain of swelling in the region of the parotid gland which is intermittent in character and painless. The existing cause of the swelling is usually trivial such as a change in temperature, a cold, chewing, etc. At times parotitis is bilateral and occasionally pus can be expressed through the orifice of the ducts. In children the parotitis may appear in the morning upon rising. Massage of the salivary glands results in the evacuation of a quantity of saliva and a reduction in the swelling. Very often an infected upper molar is found on the side of the swelling. Sialograms show the characteristic dilatation of Stensen's duct and its ramifications.

In the cases seen by the authors, the existing factor appeared to be irritation in the zone of the trigeminus and was related to dental lesions, prostheses, auricular lesions, diseased tonsils, etc. They were inclined to accept the theory that the dilatation of the salivary ducts was caused by a reflex inasmuch as there was no evidence of a mechanical obstruction.

Treatment consisted of infiltrating the sympathetics either on one or both sides with 1 or 2 per cent solution of novocaine or xilocain without adrenalin. In some cases a single injection was sufficient to relieve symptoms. Complete disappearance of symptoms was usually obtained with six or eight injections. In complicated cases in which suppuration had occurred instillation of antiseptics and antibiotic solutions into the ducts was also done.—*Murray M. Friedman, M.D.*

Larsson, Harry, and Selley, Imre. Contrast investigations on the effect of carotid-jugular fistulae in children with cerebral lesions. *Acta radiol.*, Sept., 1952, *38*, 173–386.

In 1949, Beck *et al.* described a method by which arterial blood might be supplied to the cerebral cortex via venous channels (revascularization) in children with inadequate cerebral blood supply.

By means of an anastomosis between the common carotid artery and the internal jugular vein with ligature of the vein below the anastomosis, an attempt was made to force the arterial blood to flow in a retrograde direction via the transverse sinus and the superior sagittal sinus up into the cortical veins. In this way it was hoped to obtain an increased supply of arterial blood to the capillaries in the cerebral cortex. A year later the authors stated that clinical improvement was achieved in approximately 33 per cent of their cases, while complications occurred in approximately 15 per cent.

The value of the method was investigated at the Children's Hospital, Gothenburg, in 14 cases. In association with these cases, certain angiographic examinations were carried out. In some of these the procedure was done before operation in order to judge the anatomic and physiologic conditions for operation. In other cases, it was done after operation in order to obtain, if possible, an impression of the changes which might have occurred in the circulation. These studies were supplemented by oxygen analyses of the blood. It was shown that the dynamics of the cerebral circulation were affected by the anastomosis, but the conclusion was reached that the forcing of blood into the cortical veins was not achieved.—*Mary Francis Vastine, M.D.*

Neck and Chest

Whitehouse, Walter M., and Holt, John F. Paradoxical expiratory ballooning of the hypopharynx in siblings with bilateral choanal atresia. *Radiology*, Aug., 1952, *59*, 216–220.

Two brothers with bilateral choanal atresia were studied fluoroscopically. The paradoxical ballooning of the hypopharynx that was observed during expiration was considered very striking. Normally the hypopharynx is completely or partially collapsed during expiration. This ballooning of the hypopharynx is obviously due to expiratory block of the respiratory outlets of the pharynx. The block is due to the choanal and nasopharyngeal bony atresia combined with the approximation of the soft palate and tongue on expiration. Either of these factors may be relieved by surgery, and thereby provide an air passage.

It is concluded that fluoroscopy of the pharynx is a useful procedure in following the progress of the patients who have bilateral choanal atresia.—*McClure Wilson, M.D.*

Mason, Lockert B., and Ausband, John R. Benign stenosing esophagitis associated with vomiting and intubation. *Surgery*, July, 1952, *32*, 10–16.

Esophagitis may result from exogenous corrosives such as lye and from endogenous corrosives (acid-

pepsin irritation). Whereas esophagitis is common, stenosis is rare.

In general three possible etiologic factors of esophagitis are considered. These are: (1) infection, either intraluminal (such as diphtheria) or extraluminal (such as extraesophageal lymphatic infection); (2) neurogenic factors, associated with lesions of the brain, and (3) regurgitation of gastric juice, the most commonly accepted causative factor. Experimental work shows that gastric juice has a devastating effect on the esophageal mucosa, but that it does not harm the gastric mucosa. It also has been shown that there is a frequent relationship between esophagitis and conditions associated either with vomiting or passage of a gastric tube, or both. Various views concerning the mechanism which maintains competence at the esophagogastric juncture are mentioned. General agreement on the mechanism has not been reached.

Regarding the possible role of the inlying nasogastric tube in producing esophagitis, the results of necropsy in 82 cases of acute ulcerative esophagitis are mentioned. In only 30 of these had intubation been carried out, but the lesions were no different from those in cases in which intubation had not been done. It is possible that a tube may aggravate a pre-existing esophagitis, but it is doubtful that the tube would be the primary cause of serious esophagitis.

Diagnosis of esophagitis is based on: (1) a history of dysphagia which is especially significant when associated with a history of peptic ulcer, gallbladder disease or gastric intubation; (2) evidence revealed by roentgenoscopy which is used to establish the extent of the lesion, and (3) the results of esophagoscopy and biopsy carried out to determine whether or not carcinoma is present. Most of the benign strictures and stenotic areas are found in the lower third of the esophagus. This location is thought to be in agreement with the theory of acid-peptic regurgitation.

The report of a case is presented which is interesting because of the patient's long history of intermittent vomiting without evidence of esophagitis, followed by acute and extensive stenosing esophagitis after a nasogastric tube had been left in place for three days after a biliary operation.

Treatment is carried out by direct or indirect approaches which are discussed. The direct approach, which is anatomic, includes dilatation, resection with end-to-end anastomosis or esophagogastrostomy, and plastic procedures. The indirect or physiologic approach is an attempt to reduce acid-peptic regurgitation. Subtotal gastric resection is reserved for nonmalignant cases. In the authors' case physiologic rest and neutralization of acid-pepsin were accomplished by gastrostomy and frequent feeding; an adequate pathway was established by dilatation. This combination of the direct and indirect approaches proved successful. —*André Bruwer, M.D., Ch.B.*

Rack, Frank J., Mincks, James R., and Simeone, Fiorindo A. Observations on etiology of esophageal varices. *A.M.A. Arch. Surg.*, Sept., 1952, *65*, 422–429.

A case of esophageal varices with massive hematemesis is reported in which no evidence of disease of the liver and spleen was found. Roentgen studies and esophagoscopy clearly revealed the presence of large varices in the lower third of the esophagus. The patient was explored with a porta-caval shunt in mind to relieve suspected portal hypertension. However, the liver and spleen were normal and the tensions in the portal and gastric epiploic veins were normal. A follow-up barium swallow six months later revealed a normal appearing esophagus.

Stimulated by this case the authors reviewed an additional 31 autopsy records with proved esophageal varices. In this group 22 cases had Laennec's cirrhosis or other changes characteristic of portal hypertension. In the other 9 cases with esophageal varices in the absence of cirrhosis 3 had esophageal varices associated with other abnormalities of the liver, *i.e.*, focal acute necrosis, minimal periportal fibrosis, and small metastatic nodules secondary to bronchogenic carcinoma; 5 were associated with cardiac hypertrophy and chronic passive congestion of the liver; and 1 case had esophageal varices with a normal liver and spleen. This review of cases concurred with the findings of the literature in that 80 per cent of esophageal varices was associated with Laennec's cirrhosis. The other causes of intrahepatic block of the portal system included postnecrotic cirrhosis, toxic cirrhosis, gumma of the liver, and thrombosis of the hepatic vein. The extrahepatic block of the portal venous system also caused portal hypertension and esophageal varices. Additional causes of the esophageal varices must include congenital origin, trauma and severe cardiac disease. In a small but distinct group of patients, as in the case reported, no distinct pathology can be found to account for the esophageal varices.

The authors note, as in their own case, that massive hemorrhage can occur from esophageal varices when the liver is normal and there is normal pressure in the portal vein. They have no explanation for the pathology of this condition.—*T. D. Allison, M.D.*

Kiernan, M. K. Benign intramural extramucosal tumors of the oesophagus; a report of two cases. *J. Canad. A. Radiologists*, Sept., 1952, *3*, 57–59.

Two cases of intramural and extramucosal tumors of the esophagus are reported in detail: one in a male, aged thirty-four, in which a "leiomyoma" occurred just below the notch in the esophagus produced by the arch of the aorta; the other in a male, aged forty, having a lipoma in the mid-esophageal area.

Bronchoscopic examination confirmed the location determined by the roentgen study and verified the

absence of mucosal invasion. Surgical removal was performed in each case.—*V. L. Peterson, M.D.*

Nozzoli, Franco, Epstein, Lasar, and Molnar, Ladislao. Right-sided spontaneous pneumothorax and peritoneal effusion complicating therapeutic pneumoperitoneum. *Am. Rev. Tuberc.*, July, 1952, *66*, 90–94.

Pneumoperitoneum is accompanied by complications sufficiently serious to interrupt the therapy in 5 to 6 per cent of the cases.

A single case of spontaneous pneumothorax is reported from the Sanatorium of the American Joint Distribution Committee, Merano, Italy.

The patient, a twenty-two year old man, was admitted with cavitation of the left lung uncontrolled by extrapleural pneumonolysis and bed rest. At the onset of the disease the chest roentgenogram revealed bilateral pulmonary infiltration.

Pneumoperitoneum, supplemented by left phrenic crush, was instituted. Thirteen months later and eight days after a refill of air into the peritoneal cavity a right-sided spontaneous pneumothorax occurred. Roentgenograms showed loss of peritoneal air and collapse of the right lung.

Twenty days later after complete re-expansion of the right lung a peritoneal refill was performed. A quantity of air passed from the peritoneal to the pleural space at this time. Pneumoperitoneum was effective, however, until five months later when right-sided pneumothorax again occurred with loss of abdominal air, pain in chest, and dyspnea. Removal of pleural air allowed re-expansion of the lung and pneumoperitoneum was continued.

Peritoneal effusion occurred the twenty-first month of pneumoperitoneal therapy and the procedure was discontinued.

The passage of air from the peritoneal to the pleural space indicates a communication between the spaces. Congenital variation in structure explains scrotal and mediastinal air collections in pneumoperitoneum. In the present case pneumothorax occurred, apparently, only when a high pressure was reached in the abdominal air. An opening somewhat like a hernial orifice between the two spaces could be the anomaly in this case. A right diaphragm weakened by previous inflammatory disease is also a possibility.—*Edward Julien, M.D.*

Musgrove, J. E., and MacQuigg, R. E. Successful treatment of air embolism. *J.A.M.A.*, Sept. 6, 1952, *150*, 28.

A patient observed by the authors had resective surgery for bilateral bronchiectasis. Pneumoperitoneum was chosen to aid his respiratory function because the left upper lobe failed to re-expand promptly after the second operation (left lung). After injection of 150 cc. of air under the usual precautions, the patient "appeared to have suddenly died." Precordial auscultation revealed the typical. churning "millwheel" murmur of air embolism.

About two minutes after the patient was turned on his left side, the "millwheel" murmur could not be heard. An attempt to aspirate air from the right side of the heart was unsuccessful. Other measures included lowering the head and administration of oxygen and of three ampules of 25 per cent nikethamide solution intravenously.

It was concluded that the patient's life was saved by turning him on his left side, thus relieving the "air trap" in the right ventricle. The frequent intentional use of gases in diagnosis and therapy and the occasional accidental entry of gases into the circulatory system in connection with intravenous therapy make this a timely report for all concerned. —*John W. Turner, M.D.*

Emerson, Ernest B., Jr., and Bradford, William L. Accidental inhalation of an aureomycin capsule. *A.M.A. Am. J. Dis. Child.*, Sept., 1952, *84*, 344–346.

A case of accidental inhalation of an aureomycin capsule is briefly reported in this paper. The reasons for putting this case on record as given by the authors are: (1) it afforded an unusual opportunity to observe the effects of the antibiotic, aureomycin, on the mucous membrane of the tracheobronchial tree, and (2) it will serve to point out again certain dangers inherent in capsule or tablet medication of an infant.

The patient was a nine month old white boy. Approximately thirty-six hours before his admission to the hospital, medication with an aureomycin capsule was attempted, the capsule having been included in the child's orange juice. When the child failed to take the capsule, the mother attempted to force swallowing by pushing it back over the tongue with her finger. As this was done the patient suddenly choked, coughed, and became temporarily cyanotic. At this point, one-half of the capsule was found. Wheezing and respiratory distress continued until the boy was admitted to the hospital.

Bronchoscopic examination showed severe irritative reaction of the tracheobronchial mucous membrane. The roentgenographic examination revealed increased bronchial markings but no evidence of atelectasis or emphysema. On the third day after bronchoscopy, the roentgenogram of the chest showed considerable clearing. Extensive deep bronchoscopic examination was carried out to remove as much of the yellow foreign material (aureomycin) as was possible.

The authors conclude with a plea for care when instructing mothers of infants in the art of medication and that whenever possible when dealing with a small infant some other mode of administration than capsules or tablets by the oral route be used.—*Ralph S. Bromer. M.D.*

Clagett, O. Theron, McDonald, John R., and Schmidt, Herbert W. Localized fibrous mesothelioma of the pleura. *J. Thoracic Surg.*, Sept., 1952, *24*, 213–230.

The authors report 24 cases of localized fibrous mesothelioma.

Localized fibrous mesotheliomas may arise from any portion of the visceral or parietal pleura. They must be distinguished from diffuse malignant mesotheliomas. The clinical manifestations of these tumors are largely extrathoracic and are characterized in a high percentage of cases by attacks of chills often with fever, painful and sometimes migratory joint symptoms. Clubbing of the fingers was noted in 11 of the cases. In 16 of the 24 cases joint symptoms were present.

They state that there is no characteristic or diagnostic roentgen appearance of the tumor.

The only effective treatment of these tumors is complete surgical removal. Prompt relief of the symptoms, including the extrathoracic symptoms, follows surgical removal of the tumor.—*Thomas C. Stansbury, M.D.*

Getting, Vlado A., and Lombard, Herbert L. The evaluation of pilot clinics; the mass screening or health-protection program. *New England J. Med.*, Sept. 25, 1952, *247*, 460–465.

The Massachusetts Medical Society and the State Department of Public Health jointly established four pilot clinics within the last four years for the purpose of carrying on a program of research in multiple screening. The modern concept of multiple screening consists of the application of laboratory tests by technicians to apparently well persons. Those with suspicious results are then referred to their family physicians for maximum benefit from early diagnosis and treatment. The program was designed to evaluate the sensitivity of screening tests, the motivations that brought people to the clinic, and the cost and administrative problems for a health department, hospital or private physician.

Physical examinations were performed during the first two clinic sessions in order that nearly all the positive signs resulting from the multiple screening tests and the self-screener (history form) could be evaluated. As a result of the study of findings in the first clinic, changes in procedure were made for each subsequent clinic. The third session was begun in March, 1952, and was still under way at the time of this report. The fourth session, also started in March, 1952, employed the following tests: self-screener or clinical history; blood serology, sugar and hemoglobin; urine test for sugar and albumin; vision test; hearing test; roentgen examination (4 by 5 inch film); measurement of height and weight; determination of blood pressure; hematest for occult blood; Papanicolaou test on women over thirty-five and one lead electrocardiograms. Those found to have positive results were referred to their family physicians or to an outpatient clinic for diagnosis and treatment and, the physician, in turn, was asked to report his final diagnosis.

Well over two-thirds of the patients who were asked to visit their family doctor probably did so. Statistically it was shown that approximately two-thirds of the conditions found by a combination of physical examination, laboratory tests and self-screener can be demonstrated without physical examination. The cost per person of applying the multiple screening tests, exclusive of the Papanicolaou test, ranged from $2.45 in the first session to $1.63 in the second. In a service program the use of screening tests, of modified history and overhead should cost less than $2.00 per person. Excellent tables which illustrate the various results of tests, history, physical examination and costs are presented.

The following procedures are recommended for use along with mass tuberculosis surveys.

A 70 mm. photofluorographic film of the chest for the detection of pulmonary tuberculosis, tumors and other lesions.

A blood test for detection of diabetes, anemia and syphilis.

Screening for visual defects.

A single lead electrocardiogram.

A modified self-screener or history for the detection of some other chronic diseases or verification of other test findings.

A hematest for the detection of blood in the feces may be included if desired.

Thus, it is estimated, that for about double the cost of a tuberculosis case-finding program it is possible to add the tests which will screen the same person for many additional defects.—*Daniel L. Rousseau, M.D.*

Guiss, Lewis W. Value of mass chest roentgen-ray survey methods in control of lung cancer. *Cancer*, Sept., 1952, *5*, 1035–1040.

The author presents an excellent discussion of the problems and preliminary results of screening lung neoplasm suspects from the general population as a part of a large mass chest roentgen survey. He points out that the evidence of recent reports indicates a real and serious increase in the incidence of lung cancer. The mass chest survey of asymptomatic persons is the only way to pick up cases in the silent and curable phase. When treatment is delayed until symptoms appear, more than 90 per cent of pulmonary carcinoma cases are incurable. Lung cancer has now probably replaced gastric carcinoma as the first cause of death from cancer.

As it was felt that the mass screening of chest neoplasm suspects from the general population

would accomplish nothing without prompt clinical investigation, a chest tumor registry was established. This was financed by the Department of Public Health and employed a full time secretary in addition to a consultant panel.

Status of Investigation of 3,500 Registered Cases

Chest neoplasm ruled out	1,537
Chest neoplasm confirmed	240
Lesion, metastatic	89
Goiter	417
Tumor, clinically benign not treated	206
Treatment refused	74
Lost	108
Diagnosis pending	829
	3,500

In spite of considerable effort, 14 per cent of the cases made no physician contact or refused contact with the registry and another 25 per cent are still in the process of study one year later. The author stresses that in these cases the general unawareness of physicians as to the necessity of complete investigation and immediate surgery and the ignorance of the patient as to the risk of delay are the causes for this disappointing loss of the large number of possibly curable cases.

In the 240 confirmed neoplasms 144 are pulmonary carcinomas of which 82 submitted to surgery. This is an over-all operability rate of 57 per cent. Although all the final figures are not in, approximately 8 per cent of the chest neoplasm suspects have chest neoplasms of all types.

From these findings it is recommended that a shift be made in the emphasis of cancer control education from cancer of the accessible sites to cancer of the inaccessible sites. Also, to be effective for cancer control routine chest surveys are necessary every six months. For practical purposes these can well be restricted to men over the age of forty.—*T. D. Allison, M.D.*

Bisgard, J. Dewey. Silent nontuberculous lesions of chest disclosed in survey x-ray studies. *A.M.A. Arch. Surg.*, July, 1952, *65*, 109–112.

Mass chest surveys, in addition to discovering tuberculosis, have brought to light many silent nontuberculous lesions in proportions greater than anticipated. A significant number of these lesions are malignant tumors, many remaining silent in an early stage.

The author records various series of mass examinations in this country and in England and in these the nontuberculous lesions approximated 35 per cent of the total lesions. In addition to pulmonary and mediastinal lesions, many of the listed nontuberculous abnormalities were related to the bony thorax and the cardiovascular structures.

The author believes that benign silent tumors, including tuberculosis, should be explored and as a rule resected for the following reasons:

1. They may continue to grow and produce pressure symptoms.
2. It is necessary to differentiate them from carcinoma by histopathological examination.
3. Some have a tendency to undergo carcinomatous change.
4. Tuberculomas may be a source of dissemination of the disease.—*W. W. Maver, M.D.*

Chapman, John S. Pulmonary infarction. *South. M. J.*, July, 1952, *45*, 597–602.

Although pulmonary infarction is always looked for in patients with heart failure, in those with protracted debilitating diseases, and in those in the postoperative state, the author attempts to point out that enough infarcts occur in active, apparently healthy individuals for the possibility, remote as it may seem, to be kept in mind if one is to avoid a possibly fatal outcome.

Twenty cases are presented, 2 of which were known cardiacs, with only 3 females in the entire group. Twelve of the group were active persons who had had no predisposing surgery, illness or injury.

The following tables list the clinical and roentgenographic features:

Table 1

Clinical features of pulmonary infarction

(1) Hemoptysis, at first bright red, and sometimes copious, usually followed by expectoration of dark clots and dark brown semi-liquid material.
(2) Sudden chest pain, preceding a rise in temperature.
(3) Temperature, pulse, and respiration in intermediate range.
(4) Temperature characteristically of 5 days' duration, or appearing in bouts of similar duration, unaffected by antibiotics. Tendency of temperature to return rather rapidly to normal upon heparinization.
(5) Usually moderate leukocytosis with moderate left shift.
(6) Sedimentation rate moderately elevated, with a rapid return to normal.
(7) Jaundice, if present.
(8) Electrocardiographic signs, if present.
(9) Leg signs, previous injury, previous thrombophlebitis, and so on, if present.
(10) Sterile fluid with gross or microscopic red blood cells.
(11) Freedom of sputum from pus or mucus.

TABLE II

ROENTGENOGRAPHIC FEATURES OF PULMONARY INFARCTION

(1) Peripheral location with convex or humped border toward the hilum.
(2) Long axis of infiltration parallel to longest axis of adjacent pleura.
(3) Aerated areas of tissue between adjacent areas of consolidation.
(4) Multiplicity of infiltrations and involvement of several segments.
(5) Appearance of thin, diagonally directed, intrapulmonary lines, usually within three or four days.
(6) Rapid variation of roentgenographic shadows, location, character, number.
(7) Early or immediate appearance of pleural fluid.
(8) Rapid (usually three to five days) resorption of consolidation.

The most reliable single examination consisted of satisfactory posteroanterior and lateral chest roentgenograms.

The differential diagnosis in most cases centered around pneumonia and so-called atelectasis. As far as the clinical features are concerned, the typical findings may be wanting in some cases and in others hardly any were encountered. There was no regularity.

Roentgenographically the diagnosis is on much more solid ground. There are very few forms of pneumonia which are associated with numerous, discrete and well separated areas of infiltration. Pneumonia, as a rule, does not undergo resolution within four or five days.

The variability of the roentgenographic picture from day to day may be the most useful feature, but the multiplicity of infiltrations and the very early or immediate appearance of pleural fluid are also of great importance.—*Norman Heilbrun, M.D.*

JOHNSON, RICHARD P., and JOHNSON, ELOISE E. Congenital pulmonic stenosis with open foramen ovale in infancy. *Am. Heart J.*, Sept., 1952, *44*, 344–359.

Pulmonic stenosis with open foramen ovale, though less frequent than the tetralogy of Fallot, may prove to be a common cause of the cyanotic congenital cardiac malformation. Several names have been given to the anomaly: syndrome of pulmonary stenosis with patent foramen ovale, valvular pulmonic stenosis with intact ventricular septum and patent foramen ovale, congenital pulmonic stenosis with closed ventricular septum, congenital pulmonic stenosis with intact ventricular septum, trilogy of Fallot, pulmonary stenosis without ventricular septal defect, pulmonic stenosis with reversed interatrial shunt, congenital stenosis of the pulmonic valve with associated auricular septal defect, and pure or simple isolated pulmonary stenosis.

Pathologic findings are: (1) stenosis of the pulmonic valve, the three valve cusps being fused into a single dome-shaped diaphragm, projecting into the pulmonary artery, with a tiny central perforation ranging from about 0.5 to 4 mm. in diameter. (2) The foramen ovale is open. (3) The pulmonary artery distal to the stenosis is thin-walled and dilated. (4) The right side of the heart is hypertrophied and often dilated.

The stenotic valve interferes with passage of blood from right ventricle to pulmonary artery. The right ventricle hypertrophies in response and the intraventricular pressure elevates (up to 200 mm. Hg by catheterization). Eventually the right atrium dilates, the right atrial pressure increases and when it surpasses that of the left atrium, a right to left shunt occurs. When the shunt equals about 30 per cent of the right atrial intake, cyanosis occurs.

Roentgen examination shows an enlarged heart with enlargement of the right atrium and right ventricle. Pulmonary artery is dilated but shows diminished or absent pulsations. The pulmonary vascular markings are diminished.

Cardiac catheterization demonstrates high pressure in the right ventricle but low or normal pressure in the pulmonary artery. Because of stenosis it may not be possible to pass the catheter through the valve into the pulmonary artery. When a right to left shunt is present between the atria, systemic arterial unsaturation is found.

Angiocardiography may show delay of contrast medium emptying from the right heart. The right-to-left shunt may be demonstrated by the early appearance of medium in the left atrium and aorta, but there is not the dense opacification of the aorta as seen in an overriding aorta.

The two most important malformations to be differentiated from congenital pulmonic stenosis with open foramen ovale are congenital isolated pulmonic stenosis and the tetralogy of Fallot. In the former, cyanosis is absent, except late, when it is a manifestation of cardiac failure or systemic venous stasis. In the tetralogy of Fallot the heart remains normal in size and has a "characteristic" shape, showing a concavity of the pulmonary artery segment. Other conditions to be considered in the differential diagnosis are: Ebstein's anomaly of the tricuspid valve, the Eisenmenger complex, tricuspid atresia, transposition of the great vessels, truncus arteriosus, and cor pulmonale.

Correct diagnosis is important as the usual shunt operations of the Blalock or Potts type are not helpful in this condition. Direct attack on the deformed valve by the Brock procedure may be life-saving, however.

Five cases of congenital pulmonic stenosis with open foramen ovale occurring in infants and proved by autopsy are reported.—*Harold Fulton, M.D.*

Lodge, Thomas. Cysts of the lung. *Proc. Roy. Soc. Med.*, Sept., 1952, *45*, 629–634.

The following is the classification of lung cysts according to Rizzi and de Lorenzi:

(a) Parasitic
Hydatids — Cysticercus
Pulmonary mycoses — Coccidiomycosis

(b) Congenital
A. Dermoid — F. Cholesterin cysts
B. Thymic — G. Esophageal (or gastric)
C. Branchial — H. Pericardial origin
D. Thyroid — I. Pleural origin
E. Lymphatic — J. Seromucinous cysts

Seromucinous cysts account for 23 per cent of all lung cysts, according to Schenck.

The theories for the congenital origin of pulmonary cysts can be categorized under four main headings: (1) malformation, (2) mechanical causes, (3) inflammatory causes, and (4) neoplastic origin.

Pathologically, true lung cysts consist of a fibrous outside layer, a middle layer of collagenous and elastic or muscle strands and a lining of bronchial mucosa. They contain mucinous fluid which may be gelatinous or albuminous. Increase in volume of these cysts is thought to be due, sometimes, to mucosal secretion and sometimes to cellular proliferation.

The most common parasitic cyst is the hydatid cyst. Hydatids may occur anywhere in the lung but are commoner in the right lung posteriorly. They start as round, homogeneous shadows which grow slowly and later may lose their round shape. Air may be visualized in the cyst itself or in a separate pericyst.

The most common congenital cyst is the dermoid. These are primarily found in the mediastinum anteriorly. The other congenital cysts are very rare and have no distinguishing features except the thyroid cyst which is usually mediastinal in location and tends to calcify. Lymphatic cysts are often multiple in a chain formation. Pericardial cysts may be juxtapericardial or may be some distance from the pericardium in the lung. The author has seen 3 juxtapericardial cysts, and each showed a narrow translucent layer between the cyst and the pericardium. This radiolucency is seen only when the patient is rotated and the plane is seen on end. Pericardial cysts have a predilection for the right side.

Inflammatory processes with engorgement of the lymph stations with inflammatory cells or spasm of the bronchial muscle may cause obstructive emphysematous change which may eventuate in the formation of pneumatoceles. These are circular, air-filled cavities with a sharp thin border and they sometimes contain fluid. Pneumatoceles rarely cause symptoms, but may cause cyanosis, dyspnea and cough. They are usually benign and may disappear after the resolution of the inflammatory process. The persistent cyst is typically round with a thin, hair-line circumference.

Fluid cysts are usually round or oval with a uniform density. The surrounding lung is usually normal. These cysts can simulate bronchiogenic carcinoma, and therefore should be explored in all adults. Small hamartomas can not be distinguished from fluid cysts. Similar lesions having clinical signs and symptoms which may aid in differentiating etiology are solitary metastases, tuberculomas and arteriovenous fistulas. Tuberculomas usually contain calcium, and angiography will identify the auriculoventricular fistula. Neurofibroma, intrapulmonary cysts of gastric or eosophageal origin may be difficult to differentiate. Air cysts are usually quite round and situated in the center of the lung in the paramediastinal position, although they may occur anywhere in the lung.—*Donald R. Bernhardt, M.D.*

Kane, Irving J. Segmental localization of pulmonary disease on the postero-anterior chest roentgenogram. *Radiology*, Aug., 1952, *59*, 229–237.

The author has found it possible to determine the anterior or posterior location of lung processes, as well as the segment involved, by applying the principles of roentgen-ray projection to the three dimensional anatomy of the bronchopulmonary segments.

Excellent schematic drawings demonstrate the exact normal anatomical location and shape of the five main lobes of the lungs and the more common accessory lobes.

Additional drawings demonstrate the volumetric and roentgenographic density distribution of the bronchopulmonary segments when involved with disease and as seen on the roentgenogram made in the posteroanterior projection.

The normal and diseased appearance of the bronchopulmonary segments of each lobe are discussed in detail including the factors which produce these changes.

Although this method of identification is simple and reliable it is not intended as a substitute for other views and methods of localization.—*Everett E. Seedorf, M.D.*

Parmley, Loren F., Jr., and Jones, Francis S. Primary pulmonary arteriolosclerosis. *A.M.A. Arch. Int. Med.*, Aug., 1952, *90*, 157–181.

The authors reviewed the literature on this subject and found that, although there have been over 100 cases of so-called primary pulmonary arteriolosclerosis described, less than a third have survived the critical reviews of other writers. They point out that recognition is difficult because all pathologic processes now known to cause this condition must be eliminated by detailed clinical and pathologic examination.

The theories of causation of primary pulmonary arteriolosclerosis are discussed. Most investigators have considered that pulmonary hypertension was the mechanism responsible and likened the arteriolar changes to those found in the arteriolonephrosclerosis of systemic hypertension. However, it is pointed out that there are many instances of pulmonary arteriolosclerosis in which hypertension is known not to exist. Cardiac catheterization has made it possible to substantiate this by direct measures. Some have considered the cause to be a developmental defect of the arterioles. The role of hypoxia in the production of arteriolosclerosis is discussed.

The clinical picture and pathology are described, and charts of clinical and pathological data on authentic cases in the literature are presented. Case reports are made on 3 patients of the authors, 1 of which was diagnosed during life by exploratory thoracotomy and biopsy. Roentgenographically the earliest indication of this disease is that of an accentuated pulmonary conus-artery segment. Later, right ventricular hypertrophy may develop.

In summary, 28 cases of primary pulmonary arteriolosclerosis collected from the literature are presented, to which are added the authors' 3 additional cases.—*D. S. Kellogg, M.D.*

Scher, Jordan M., and Jaruszewski, Edward. Virus influenza A′ infection with pulmonary manifestations. *A.M.A. Arch. Int. Med.*, Aug., 1952, *90*, 201–216.

This is a report on 76 patients with clinical influenza admitted to the U. S. Naval Hospital, Philadelphia, with a recording of symptoms, physical findings, and the treatment used. The diagnosis of influenza virus infection was accepted when the following criteria were met: (1) presence of serological evidence of recent infection by the influenza virus; (2) demonstration of a clinical course compatible with such infection; (3) absence of bacterial pathogens as etiological agents.

About 31 per cent of the patients showed infiltration in the roentgenograms. Dividing each lung into three sectors, it was found that the involvement was predominantly in the lower sectors. About a third of these involved the costophrenic angles, tentatively localized to the posterior basal segments. The upper sectors showed no involvement. It was noted that the administration of antibiotics did not greatly affect the presence of the infiltrations, which persisted an average of 13.4 days in those treated and 15.1 days in those untreated.

Four case histories are presented, with reproductions of roentgenograms.—*D. S. Kellogg, M.D.*

Siddons, A. H. M., and MacArthur A. M. Carcinomata developing at the site of foreign bodies in the lung. *Brit. J. Surg.*, May, 1952, *39*, 542–545.

This paper records 2 cases of carcinoma of the bronchus occurring at the site of retained metallic foreign bodies. In both cases the foreign bodies were war missiles and the patients died of carcinoma thirty-two and thirty-five years after injury. In 1 case the diagnosis was not proven histopathologically, although the bronchoscopic findings and clinical course strongly support the diagnosis.

Brief summaries of 5 other cases obtained from the literature are reviewed in which malignant growths were diagnosed at the site of retained foreign bodies which had been present from six to thirty-six years.

In 4 of the 7 cases there was associated abscess formation and in another bronchiectasis. This might suggest that the causal factor in producing carcinoma was prolonged sepsis rather than the presence of the foreign body. However, the authors do not support this view and state that the sequence of events in several cases suggests that the sepsis which developed was itself the result of bronchial obstruction by neoplasm.—*Paul R. Dirkse, M.D.*

Rossi, Lucio. La stratigrafia nello studio delle localizzazioni mediastino-ilari nella tubercolosi infantile. (Stratigraphy in the study of localization of mediastinal hilar lymph nodes in childhood tuberculosis.) *Ann. radiol. diag.*, 1952, *24*, 101–160.

The limitations of conventional roentgenograms of the chest are set forth in the study of childhood tuberculosis because of the numerous organs and structures contained within the mediastinum. To explore more thoroughly the hilar and mediastinal changes in these cases, the author has resorted to laminagraphy. The technique for taking these films is described. Most of the patients reported in this article are two years of age or older. In certain cases stereolaminagraphy was utilized to advantage.

The normal anatomical relationship of the lungs and mediastinal structures, as seen at the various levels are described in detail.

Case reports are listed of childhood tuberculosis in the latent stage, as well as questionable cases or where the primary focus of infection remained obscure. Also cases of tracheobronchial adenopathy were studied and others which had parahilar, paratracheal and tracheobronchial adenopathy. In some cases by using this method, one could differentiate mitral heart disease from mediastinal adenopathy. Lesions of the lung parenchyma and stenosis of bronchi could be studied in detail in cases of active lung tuberculosis.

The author concludes that supplemental laminagraphy is indispensable to the study of lung diseases in the field of pediatrics. By this method glandular tuberculosis can be studied with accurate visualization, location and extent of involvement. Latent or obscure cases can be detected and the extent of the parenchymal lesions likewise can be better visualized

and evaluated. In the more difficult cases stereolaminagraphy is recommended. The only limitation the author sees for use of this method is in children less than one year of age. This could be overcome by faster technique.

This is an excellent article containing considerable detailed information on chest laminagrams. Numerous cases with good illustrations are included. Obviously the method is useful in the study of any mediastinal pathology, including tuberculosis.—*Peter E. Russo, M.D.*

LENTINI, SIRIO. Tomography applied to the posterior pneumo-mediastinum in the diagnosis of diseases of the mediastinum. *Scientia Med. Ital.*, 1951, *2*, 191–238.

The author applied this new technique (pneumomediastinal laminagraphy) to the anterior mediastinum and obtained important results which may be summarized as follows: (1) visualization of the thymus both in lateral and sagittal projection in normal adults; (2) the possibility of differentiating the thymus from its residues of fatty connective tissue and to achieve their separation from the shadow of the large vessels; (3) visualization of the large veins (superior cava, the brachiocephalic veins and in lateral position the azygous vein) as well as the arteries branching off from the aorta.

The laminagraphic method, being highly selective, obviously has great advantages for the study of the mediastinum. When associated with pneumomediastinum, the advantages of both methods are added while the disadvantages cancel each other. In all cases examined with posterior pneumomediastinum a small amount of gas penetrates into the anterior mediastinal space, thus rendering possible the exploration of the whole mediastinum.

Posterior pneumomediastinum normally always yields a constant roentgenological picture, so that any anomaly in the distribution of the gas must always be attributed to a pathological condition. It is performed by the author according to Condorelli's technique which consists essentially of a transtracheal puncture just above the jugulum using a long thin needle and allowing gas (oxygen or air) to enter the mediastinal space between the trachea and the esophagus.

The lesions examined by posterior pneumomediastinal laminagraphy include: (1) diseases of the blood vessels, (a) affections of the large mediastinal blood vessels and in particular the aorta, (b) congenital heart disease, (2) neoplasms and cysts, (a) tumors and cysts of the mediastinum, (b) metastases and invasions of pulmonary tumors.

Other roentgenological methods, such as angiocardiography, bronchography and the passage of barium through the esophagus certainly yield clearer results for the individual organs. However, they distort other organs which are not under examination, and can only furnish indirect information about these. The advantage of pneumomediastinal laminagraphy is, of course, that all the mediastinal organs can be studied simultaneously in their respective relationship to each other.

When the diagnostic problem is concerned with only one organ, the use of these selective methods is justified. In all other cases, the author believes the laminagraphic examination associated with pneumomediastinum should be done first. When necessary, it can conveniently be followed by one of the special methods, particularly as the gas introduced into the mediastinum stays there for more than twenty-four hours.

The ordinary laminagraphic visibility of the aorta, both in sagittal and lateral projection, is confined to the arch. The ascending part remains obscure while the descending part is hardly ever visible. When laminagraphy is combined with posterior pneumomediastinum, the aorta can be explored in its whole length and its main branches appear so clearly that their course and their caliber can be examined. The pulmonary blood vessels, particularly the artery, its bifurcation and its branches are visualized on plain laminagrams, but here again they stand out much more clearly when using posterior pneumomediastinal laminagraphy. The method requires only one operator and modest equipment. It is well tolerated, indeed it can be done ambulatorily. Angiocardiography, on the other hand, is a far more complicated procedure, requiring a considerable amount of equipment and precautions. It is not without danger in luetics, arterial hypotension and in cases of myocardial insufficiency.

In tumors of the lung, posterior pneumomediastinal laminagraphy permits a simultaneous investigation of all the problems concerning the mediastinum, which are: (1) the presence of lymph node metastases and their localization; (2) the vascular features and their relation to the tumor and its metastases; (3) the features of the trachea, the large bronchi and the bifurcation, either as regards the possible presence of lymph node metastases or as regards neoplastic destruction.

The indications of pneumomediastinal laminagraphy are essentially the same as those for plain laminagraphy.

In diseases of mediastinal blood vessels, posterior pneumomediastinal laminagraphy can contribute to the solving of many diagnostic problems as is shown in cases of syphilitic aortitis and of aortic aneurysm, which are discussed in detail.

In congenital angiocardiopathies, the pneumomediastinum makes it possible to differentiate the extracardiac lesions from the complex malformations of the blood vessels and heart as is shown in cases of aortic coarctation and of Fallot's tetralogy.

In the diagnosis of tumors or cysts of the mediastinum, posterior pneumomediastinal laminagraphy has its special and most important application and

would appear to be the roentgen diagnostic method of choice.—*Stephen N. Tager, M.D.*

Pettinari, Vittorio, and Baccaglini, Mario. The pneumomediastinum: technique and findings. *Scientia Med. Ital.*, 1951, *2*, 239–268.

Pneumomediastinum is an important method of roentgenological investigation of the affections of the thorax, the possibilities of which have not been sufficiently explored.

The insufflation of gas via the presacral route has the great advantage of being easily performed and of not causing inconvenience. It makes all the necessary movements of the patient possible, even while the gas is being introduced. Spreading between the various organs, the gas occupies a series of spaces which from below upwards are the following: (1) the retro-aortic space; (2) the aorto-pericardial space (including the esophagus); (3) the pericardio-diaphragmatic space; (4) the cardio-costo-phrenic space; (5) the pericardio-sternal space. Apart from these large spaces the gas collects in important smaller areas such as the area between the aortic arch and the hilus of the left lung, the area between the aortic arch, the hili of the lungs and the pulmonary arteries, the area around the vena azygos and the peritracheal region.

Two other procedures are also being used: insufflation via the jugular-sternal route for the anterior mediastinum and insufflation via the paratracheal route for posterior mediastinum (Condorelli).

The presacral technique is described in great detail.

Pneumomediastinum is the only method today available which permits a clear visualization of the thymic region.

Malignant tumors with an extracapsular development appear in the form of an irregular mass with no clear outline, which can obstruct or render more difficult the passage of the gas.

The pericardium appears in its entirety and indirectly outlines the heart shadow. Every change can therefore be shown and in the case of tumors originating from it, this method reveals the point of origin. This clarifies the differential diagnosis which is otherwise difficult, particularly as against neurinomas and other mediastinal lesions. In a case of pericardial sarcoma, pneumomediastinum made possible an exact diagnosis confirmed by operation.

In a case of stenosis of the aortic isthmus, pneumomediastinum showed how the profile of the blood vessel was markedly narrowed below the arc, while the subclavian had a concave external profile. The subsequent angiocardiographic control presented an exactly identical picture.

In the case of Fallot's disease, pneumomediastinum revealed the unification of the aortopulmonary trunk, while the pulmonary artery was only separated by a thin layer of gas in the upper part. Subsequent angiocardiographic control confirmed the simultaneous filling of both vessels.

In a patient with a retroclavicular pulsating swelling, it was easy to demonstrate an aneurysm of the carotid.

Pneumomediastinum has a definite place in the diagnosis of cardiovascular affections. In affections of the esophagus, pneumomediastinum chiefly concerns tumors.

The value of the method lies especially in the information it yields about the relationship of a tumor mass with the neighboring organs. Indeed it furnishes useful indications on the adhesions of the neoplasm and thus on its operability. If the tumor is free and not too voluminous, the gas surrounds it completely, or for a good part, and is then able to continue moving upwards. *When, on the other hand, the growth has already spread into the periesophageal tissues, the gas is stopped at that point.*

Pneumomediastinum is useful in pulmonary tumors, since it outlines the spread into the hilus and the mediastinum. When large masses in this region are present, *there is either a partial or total block and the gas cannot spread. This is often a sign that the growth has exceeded the limits of operability.*

Pneumomediastinum via the presacral route requires only a relatively small amount of gas which diffuses into the mediastinum along physiological paths and does not, therefore, produce artefacts occurring with other techniques which employ the direct introduction of gas into the mediastinal spaces under considerable pressure.—*Stephen N. Tager, M.D.*

Burchell, Howard B., and Pugh, David G. Uncomplicated isolated dextrocardia ("dextroversio cordis" type). *Am. Heart J.*, Aug., 1952, *44*, 196–206.

Uncomplicated isolated dextrocardia implies dextrocardia without inversion of the cardiac chambers and normal position of the intra-abdominal organs.

In the past fifteen years, 12 instances of isolated dextrocardia have been studied at the Mayo Clinic. These 12 cases were contained in a total of approximately 100 cases of dextrocardia. This report concerns the 5 cases in which there was no discernible abnormality of cardiac function; these cases showed right-sided hearts without situs inversus abdominalis, the aortic arch and descending aorta on the left, and the pulmonary arterial bifurcation occupying its normal position to the left of the aorta. These relationships were clearly demonstrated in an angiocardiogram on 1 patient.

Because of the lack of inversion of the cardiac chambers, the electrocardiographic tracings obtained on these patients may deviate remarkably little from normal.

Due to some confusion of terminology in describing these cases, the alternative simple term "dextro-

rotation of the heart" is proposed by the authors to describe cases which fulfill the following criteria: (1) the great vessels have a normal relationship at the heart base, (2) the heart is normal functionally, and (3) inversion of cardiac chambers is absent.—*Harold Fulton, M.D.*

Kirklin, John W., Burchell, Howard B., Pugh, David G., Burke, Edmund C., and Mills, Stephen D. Surgical treatment of coarctation of the aorta in a ten week old infant; report of a case. *Circulation*, Sept., 1952, *6*, 411–414.

This is a report of an interesting case in which a surgical correction of coarctation of the aorta was carried out successfully in a ten week old infant. The authors state that the optimal age for surgical treatment for coarctation of the aorta is between the ages of ten and twelve years but when cardiac failure is imminent surgical treatment should not be withheld even in infancy.

Coarctation of the aorta must be ruled out in unexplained cases of cardiac enlargement in infancy. As it is possible to detect presence or absence of pulsations in the femoral vessels after the fifth day, a clinical diagnosis can be made this early. The precise method for determining this malformation is aortography. The differential diagnosis in such cases includes patent ductus arteriosus and endocardial sclerosis of the large ventricle type.

Follow up examinations show the child's growth and development to be essentially normal. When last seen at the age of eighteen months, the size of the heart had returned to normal.—*T. D. Allison, M.D.*

Ruskin, Herman, and Samuel, Eric. Rheumatic heart disease with calcification of the left auricle; report of two cases with review of the literature. *Am. Heart J.*, Sept., 1952, *44*, 333–343.

Calcification in the left auricular (atrial) wall is rare and in the majority of recorded instances it has been associated with rheumatic heart disease involving the mitral valve. Six cases from the literature, diagnosed during life, have been examined postmortem. In every case the calcification was endocardial, the extent varying from plaques in the posterior wall to involvement of the entire wall. In all cases there was mitral stenosis with varying degrees of incompetence.

The roentgen features are well established. The calcification is generally curvilinear in character and occupies a position posterior to the base of the heart. As it is associated with mitral valve disease, some enlargement of the left atrium is usually present and consequently the arc of calcification is larger than one would anticipate with a normal sized left atrium. Such calcification may not be seen in the routine posteroanterior view, but is usually obvious in the right anterior oblique projection where its relation to the displaced barium-filled esophagus can be clearly demonstrated.

Calcification in other cardiac structures, such as pericardium, ventricular myocardium, coronary arteries, annulus fibrosus, and valves, are not likely to be confused with left atrial calcification. Very occasionally a mural thrombus in the left auricle becomes calcified, but massive thrombi do not calcify.

The clinical course of patients with calcification of the left atrial wall secondary to mitral disease is striking in that the patients live relatively longer than those patients with similar valvular involvement without the atrial calcification.

Two cases are discussed and illustrated by roentgenograms.—*Harold Fulton, M.D.*

Vesell, Harry, and Schack, Jerome A. Schistosomal heart disease; bilharzic cor pulmonale. *Am. Heart J.*, Aug., 1952, *44*, 296–305.

Infrequently schistosomiasis may involve, indirectly, the heart, producing a striking clinical and pathological cardiac entity. The first reported case of schistosomal heart disease diagnosed ante mortem in the United States is presented.

A thirty year old white male who for the last twenty years of his life lived in Israel complained of dyspnea on exertion. Roentgenographic study relealed a moderately enlarged heart and extreme enlargement of the major pulmonary vessels which exhibited normal amplitude of pulsation. The roentgen appearance was compatible with that of an atrial septal defect, but angiocardiography showed no evidence of interatrial shunt.

Laboratory examination of the urine showed many erythrocytes, and eventually schistosomal eggs were demonstrated in concentrated urine specimens.

The patient died shortly thereafter: postmortem examination was not done, but the authors believe that the clinical, laboratory, and roentgenologic findings in the case are sufficiently characteristic to make the diagnosis of schistosomal heart disease.—*Harold Fulton, M.D.*

Abdomen

Seedorf, E. E., Powell, W. N., Greenlee, R. G., and Dysart, D. N. Pseudo-albuminuria following Monophen and Priodax. *Radiology*, Sept., 1952, *59*, 422–423.

The authors report the incidence of pseudo-albuminuria in patients following the ingestion of monophen and priodax. Monophen appeared to produce a slightly higher incidence of "false" albuminuria, but it was less prone to produce undesirable side reactions than priodax. The incidence of pseudo-

albuminuria is high when Robert's reagent or the nitric acid and heat test is employed.

Both drugs produced comparable good cholecystograms.—*Dean Nichols, M.D.*

Harrington, Leo A., Schwinger, Harold N., and Schwinger, Aaron. The significance of the post-fat cholecystogram. *Am. J. Digest. Dis.*, Sept., 1952, *19*, 284–286.

The authors felt that there was still need for basic research regarding motor activity of the gallbladder if one is to judge by the conflicting opinions regarding delayed emptying of the biliary viscus.

The differences of opinion regarding the importance of the post-fat cholecystogram are cited with little discussion. Their report resulting from a study of 100 patients with no history of biliary disease is presented. They found that the gallbladder contracted to some degree in 99 per cent as shown on roentgenograms made twenty-five and forty-five minutes following a fat meal. In convalescent patients of nonbiliary disease also good contraction of the gallbladder was found. Factors of race, sex, weight, and age are tabulated.

From this work the authors, by inference, place considerable weight upon the post-fat roentgenograms in the evaluation of cholecystographic examination.—*Robert D. Moreton, M.D.*

Schrems, H. Röntgenschichtuntersuchung der Gallenblase. (Laminagraphic examination of the gallbladder.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Sept., 1952, *77*, 322–330.

Following the example of several European and American investigators, the author has used laminagraphy for the examination of the contrast-filled gallbladder

Exposures are made at 2.5 to 6.0 cm. depth, with the patient supine.

Especially in the presence of superimposed gas or other extrinsic substances, laminagraphy is useful for the demonstration of stones and other abnormalities. The method is not used routinely but only in those instances in which conventional techniques fail to yield an adequate result.

The article is well illustrated with reproductions of a series of pertinent roentgenograms.—*Albert Oppenheimer, M.D.*

Järvinen, Klaus A. J., and Paltia, Vilho E. Differential diagnosis of hepatitis and liver cirrhosis by means of cholecystography. *Am. J. Digest. Dis.*, Aug., 1952, *19*, 244–248.

Soon after the discovery of cholecystography, the relationship of liver function and cholecystography was studied with the conclusion that it is unsuccessful in connection with liver cirrhosis. In recent years with the development of additional liver tests, the authors thought it would be beneficial again to correlate this roentgen examination with different laboratory studies.

Others had demonstrated that in the use of bromsulphthalein, if the retention is above 40 per cent, cholecystography is unsatisfactory; if it is 30 to 40 per cent it is uncertain; and if it remains below 30 per cent it is usually satisfactory. This is to be expected because of the physical and chemical similarity of the contrast media used in cholecystography and in the bromsulphthalein tests. Both tests are of no value when jaundice is severe.

The authors' study included cholecystography in 23 patients with hepatitis and 23 patients with liver cirrhosis. The studies were made only if the icteric index was 1:30 or below. In the cases of liver cirrhosis, no normal cholecystography was observed, and almost 70 per cent of the gallbladders were not distinctly visualized. On the other hand, among the 23 cases of hepatitis only one gallbladder was not visualized and in this patient an associated cholelithiasis was also present. Cholecystography in the great majority of cases of hepatitis was normal.

The authors attempted to correlate the findings of cholecystography with liver function tests, such as the Takata-Ara, mercuric chloride, thymol turbidity, zinc sulfate turbidity, alkaline phosphatase and the albumin-globulin ratio. A definite and direct correlation was present between cholecystography and the Takata-Ara test. It is their feeling that there will also be some correlation with the other function tests, not so well defined or obvious. Further study would be necessary to determine this.

The role of cholecystography in the study of liver function has been more or less neglected in recent years. It is the authors' contention that in more isolated areas and in regions where facilities are not available, cholecystography may give important information differentiating chiefly between hepatitis and hepatic cirrhosis, in the former most often being normal, and in the latter being distinctly abnormal. They consider nonvisualization of the gallbladder suggestive of liver cirrhosis if the contrast medium is taken properly, if affections of the gallbladder have been taken into account, and if other systemic diseases such as pernicious anemia, exophthalmic goiter, and diabetes mellitus have been excluded.—*Robert L. Raphael, M.D.*

Sachs, Allan E. Absence of common bile duct. *J.A.M.A.*, Aug. 16, 1952, *149*, 1462–1463.

Anomalies of the biliary ducts are a constant source of concern to surgeons performing operation in that area. Numerous variations are reported as to position, length, and even aberrant sites.

Absence of the common bile duct has been described briefly but once before. This anatomic

variation will be recognized, as it was in the case reported here, only if the helpful practice of tracing the biliary ducts from within after the gallbladder is emptied is employed.

A single case report is presented of a fifty-four year old school teacher with the history of severe acute pain in the right upper abdomen accompanied by nausea and vomiting. Diarrhea developed, the stools became clay colored and jaundice and pruritus were noted.

Cholecystograms done two years previously for flatulence and constipation showed no concentration of the contrast medium.

At operation, an inflamed, dilated gallbladder was found and stones were palpated in the cystic duct. The cystic duct was shown, by probing, to enter the duodenum with absence of the common duct. The hepatic ducts entered the gallbladder in its midportion. A cholecystotomy was performed. A cholangiogram subsequently outlined the anomaly clearly.

A review of the literature is given and the possible mechanisms of development of an absent common duct are discussed.—*Lawrence R. James, M.D.*

TWISS, J. RUSSELL, and CARTER, R. FRANKLIN. The relationship of biliary tract disorders to diabetes mellitus. *Am. J. M. Sc.*, Sept., 1952, *224*, 263–273.

Diabetic patients have long been known to have a high incidence of biliary tract disorders. It is the purpose of this article to review and to illustrate how diabetes mellitus may be caused or aggravated by disorders of the biliary tract. The authors discuss (a) the anatomic and physiologic relationships of the biliary tract and the pancreas and (b) the effect of functional disorders and organic disease of the biliary tract upon the pancreas. An accumulated clinical experience now justifies conclusions as to how an adequate management of such conditions may have therapeutic value for the diabetic patient.

A review of the literature revealed the following facts: (a) Cholecystographic evidence of gallbladder dysfunction was found in 44 per cent of a series of 70 diabetics over forty years of age. (b) In an analysis of patients with gallbladder disease there were nine times as many who had diabetes as would be expected. (c) Incidence of diabetes in cholecystitis was greater than in cholelithiasis. (d) In acute pancreatitis the incidence of diabetes was forty times that of chance. (e) Cholelithiasis has been found at autopsy in 31 per cent of diabetics with an incidence of 21 per cent in a controlled series of nondiabetics. (f) Infection of the biliary tract is a common precursor of the pancreatitis which sometimes precedes diabetes mellitus, and the onset of the gallbladder disease is usually earlier than that of diabetes. (g) In adults, cholelithiasis is probably one of the most important etiologic factors in diabetes mellitus.

Significant anatomic features are: (a) the path of the common bile duct through the head of the pancreas, and (b) the common opening of the pancreatic and common bile ducts above the sphincter of Oddi. Hjorth, on the basis of anatomic cholangiographic studies in a large series coming to autopsy, states that 80 per cent show a common opening of this type. This common opening may permit a single lesion to block both ducts, resulting in pathologic changes in either or both systems.

Associated physiologic disturbances have been found to occur in the pancreas as the result of biliary tract disorders. Thus in a quoted series of 115 patients with chronic gallbladder disease having symptoms suggestive of pancreatic disease, the majority showed deficiencies in the external secretion of amylase, lipase and protease. There was also evidence of a disturbed internal pancreatic secretion in that half of the patients showed a hyperglycemic and half a hypoglycemic tendency.

Spasm of the common duct sphincter may produce (a) biliary stasis with a predisposition to activation of an existing infection, (b) progressive infectious diseases of the bile ducts with ultimately liver and pancreatic involvement, (c) stone formation with an additional obstructive element.

In patients with a common opening of the pancreatic and common bile ducts, an obstructive lesion of the ampulla may lead to (a) biliary reflux of pancreatic secretion into the bile ducts and gallbladder, (b) pancreatic reflux of bile into the pancreatic ducts, and (c) pancreatitis with possibly a subsequent diabetes.

The associated pathologic conditions of the biliary tract which may precede or accompany diabetes are: sphincter spasm, infectious cholecystitis, cholangitis, cholelithiasis, cancer of the gastrointestinal tract, and a benign or malignant lesion of the biliary tract. Concomitant liver changes may be hepatitis, fatty infiltration of the liver or portal cirrhosis. Hyperglycemia, but not glycosuria, was found in 80 per cent of one series of patients with cholelithiasis.

Surgical Considerations. The presence in the diabetic patient of organic infection or obstructive biliary tract disease is an indication for surgery. Early recognition and treatment of these conditions may prevent unfavorable progression of the biliary tract disease, and also diminish the likelihood of complications such as acute cholecystitis and pancreatitis; furthermore, surgical correction of such conditions may lessen the severity of the diabetes.—*Robert K. Arbuckle, M.D.*

ARNHEIM, ERNEST E. Congenital hernia of the diaphragm with special reference to right-sided hernia of the liver and intestines. *Surg., Gynec. & Obst.*, Sept., 1952, *95*, 293–307.

Failure of fusion of the various developmental portions of the diaphragm or failure of muscular de-

velopment in these regions results in congenital hernia of the diaphragm. The most common site of developmental abnormality of the diaphragm is in its posterior portion. The identity of location and relations of the pleuroperitoneal opening in the fetus and the hernial opening suggest that a persistent pleuroperitoneal hiatus is the developmental cause. The location of this defect in the costovertebral area has become associated with the name "foramen of Bochdalek." Not infrequently the posterior defect in the diaphragm is large and is thought to result from failure of formation of that portion of the diaphragm derived from the pleuroperitoneal membrane; it occupies the posterolateral portion of the diaphragm and usually extends posteriorly from the eighth rib and medially to the esophageal hiatus.

These types of hernia are much more common on the left side than the right. From an embryologic study this is explained by the earlier closure of the right pleuroperitoneal membrane and the protection of the liver.

Most congenital diaphragmatic hernias do not have a hernial sac and there is a free communication between the pleural cavity and the abdomen ("false hernias"). The sac is a thin membrane which is usually attached to a cresent-shaped shelf of diaphragm laterally, a segment of diaphragm medially, and is reflected over the viscera in the thorax as a hood.

The most common abdominal viscera involved in congenital diaphragmatic hernia are the small intestine and the ascending and transverse colon, *i.e.*, that portion of the intestinal tract derived from the midgut in the fetus. Occasionally the stomach is herniated, and either the spleen or liver, according to the side of the defect.

These herniations are probably present by the fourth month of fetal life inasmuch as the pleuroperitoneal foramina are usually closed by this time. It is remarkable that fusion between the herniated intestines and the pleura does not occur. Adhesions between the herniated viscera and the intrathoracic surfaces are rarely found.

The displaced viscera in the thorax compress the homolateral lung which is usually found as an airless mass of dark red tissue compressed against the apex of the chest. The heart and mediastinal structures are displaced to the opposite side. The small size of the peritoneal cavity results from the absence of the herniated viscera.

Prompt surgical treatment of congenital diaphragmatic hernia is extremly important in the newborn period. A review of 30 newborn infants with this condition showed the duration of life to be less than one hour in 13, less than twenty-four hours in 9, and less than thirty-six hours in 2 patients.

The author reports 3 cases of congenital right-sided diaphragmatic hernia of the liver and intestines successfully treated surgically.—*Mary Frances Vastine, M.D.*

Capurro, Federico Garcia, and Pecantet, Juan Carlos. A new technic in the study of the mucosal pattern in the cardiac area of the stomach. *Radiology*, Aug., 1952, *59*, 198–200.

A new method is offered for visualization of the mucosa of the fundus of the stomach. Either by aspirating the gas bubble with a duodenal tube or by pneumoperitoneum, the fundus is reduced to a spindle-shaped segment much like the esophagus. When 10–15 cc. of barium is injected through the tube, it spreads in a thin layer between the folds of the collapsed gastric fundus, demonstrating the mucosal detail. Pneumoperitoneum also is useful for the diagnosis of lesions growing out of the stomach and for differentiation of extragastric lesions. The two techniques are often used in combination.—*William A. Goodrich, M.D.*

Rappaport, Emanuel M., Rappaport, Eugene O., and Alper, Abe. Incidence and clinical significance of transpyloric prolapse of gastric mucosa. *J.A.M.A.*, Sept. 20, 1952, *150*, 182–188.

The authors briefly review the literature and the divergence of opinions relative to transpyloric prolapse of the gastric mucosa, reporting on a series of 1,000 consecutive roentgenological studies of the upper gastrointestinal tract of their own. Because of the transient nature of the prolapse their roentgen criteria of the herniation of the gastric mucosa through the pylorus are defined as the demonstration on any single film of the duodenal cap region of a "mushroom or umbrella shaped defect of the base of the duodenal bulb with lobulation caused by the antral folds."

Prolapse was found in their series in 15.5 per cent of cases studied. Occurrence was much more frequent in hypersthenic persons with transverse stomachs and slightly more frequent in men. The finding was more common over the age of thirty. Extent of the herniation was usually small and no appreciable relationship of extent and upper gastrointestinal symptoms was found. Prolapse was noted in 10.7 per cent of persons without symptoms, in 13.6 per cent of persons with lower bowel symptoms and in 15.9 per cent of persons with upper digestive tract symptoms.

Transpyloric prolapse was found associated with every common disease of the gastrointestinal tract, the highest incidence of association being 24.1 per cent with hiatus hernia. Gastric ulcer patients showed 15.8 per cent and duodenal ulcer patients 15.6 per cent associated prolapse; 19 per cent of patients with proved gastritis and 18.5 per cent of patients with cholecystitis and/or cholelithiasis revealed this finding. Other conditions showed a lower incidence of associated prolapse. The authors do not feel that the incidence of prolapse with proved gastritis was sig-

nificantly greater than other associated conditions to implicate it as an etiological factor.

Relative to the improvement of symptoms in patients with associated diseases, the authors found that there was little difference in the degree of improvement of symptoms after therapy in patients with associated prolapse as compared to those with a single finding without prolapse. When transpyloric prolapse of the gastric mucosa occurs without associated disease the symptoms are chiefly functional.

The authors stress that all other symptom producing factors must be excluded before implicating prolapsed gastric mucosa as the causative agent.—*C. E. Sherwood, M.D.*

Littner, Michael, and Kirsh, Israel. Aberrant pancreatic tissue in the gastric antrum; report of seven cases. *Radiology*, Aug., 1952, *59*, 201–211.

The authors review the literature of this unusual lesion and report 7 cases. In some instances, it should be possible to suggest the presence of the condition from the roentgen examination.

The following characteristic findings may be present: (1) there is a nodular filling defect about 1-2 cm. in its greatest diameter, usually located in the gastric antrum; (2) the shape of the lesion varies from round to oval and is sharply demarcated from the surrounding normal stomach; (3) a central pit indicated by a barium fleck may be seen; (4) the peristalsis is normal and there is absence of spasm, palpable mass or gastric residue.

The signs and symptoms, if present, are not diagnostic and may mimic peptic ulcer, cholecystitis, or biliary colic.

Lesions to be considered in the differential diagnosis are polyps, lymphoma, intramural neoplasm, carcinoma of the stomach and gastric ulcer.—*McClure Wilson, M.D.*

Barbosa, Jorge de Castro; de Castro, V. Bicudo; Caminha, Nicola; de Oliveira, Candido, and Ramos, Carlos. Duplication of the stomach; report of the fifth case recorded. *J.A.M.A.*, Aug. 23, 1952, *149*, 1552–1555.

The authors report the fifth recorded case of duplication of the stomach. Lesions of this type have been found at all levels of the digestive tract. In 1940 Ladd and Gross reported the first case of duplication of the stomach. In 1951 Hoyer and Adresen reported a communicating duct between the gastric cardia and the duodenal bulb. In a series of 18 cases collected by Ferber almost every level of the gastrointestinal tract was represented.

Pathologically, the lining of the duplicated areas is formed of mucosa similar to that of some portion of the digestive tract. The duplication is not necessarily a replica of the viscus to which it is contiguous. Communication between the viscus and the duplication is not usual but when present allows the passage of digestive contents into the cyst. The differentiation between duplication and lymphatic mesenteric cysts is of practical surgical importance. While cysts can be shelled out easily from the mesentery there is no plane of cleavage in the case of duplication.

The symptoms caused by duplications of the digestive tract arise from obstruction of the lumen, distention of the hollow structure and vascular obstruction.

While roentgenograms will not disclose the histopathological nature of the mass, the use of contrast material may be of value in determining that the mass is intramural and extramucosal. The treatment of duplication is primarily surgical.

The authors' case, a woman, aged thirty-three, was admitted complaining of epigastric pain, vomiting and constipation. Examination revealed a left abdominal mass which had gradually increased in size over a period of four months. Roentgenological study of she stomach showed a voluminous extragastric tumor mass that displaced the stomach forward. The impression was that of a large pancreatic cyst.

At operation a thick-walled cystic mass was found adherent to the posterior surface and greater curvature of the stomach and to the anterior surfaces of the pancreas and duodenum. More than 2,000 cc. of clear, light yellow fluid of neutral reaction was aspirated. A partial hemispherical excision of the cyst followed by its marsupialization to the anterior abdominal wall was completed after painting the lining with 10 per cent solution of formaldehyde.—*J. N. Ané, M.D.*

Gottlieb, Charles, Beranbaum, Samuel L., and Weiner, Melvyn L. Syphilis of the stomach. *Radiology*, Aug., 1952, *59*, 193–197.

Two cases of gastric syphilis are reported: One was diagnosed by operation and autopsy, and the other by complete disappearance of the lesion following specific therapy. Most of the cases in the literature have been diagnosed clinically. The accepted criteria for such a diagnosis are: (1) untreated tertiary syphilis, (2) a demonstrable roentgen defect, (3) presence of gastric symptoms, (4) no change by usual medical treatment, (5) symptom relief and disappearance of defect after specific therapy.

In 70 per cent of cases, the defects seen fluoroscopically occur in the prepyloric region. An hourglass deformity is found in 22 per cent and there is diffuse involvement in 8 per cent. The symptoms may suggest decreased gastric capacity, cancer, or ulcer. The histology of the gumma is discussed.—*William A. Goodrich, M.D.*

Gagliardi, Raymond A. Preoperative roentgen demonstration of non-opaque foreign body

in the duodenum; report of case. *Radiology*, Sept., 1952, *59*, 419–421.

Nonopaque foreign bodies in the gastrointestinal tract, if they manifest themselves at all, usually do so by production of an abdominal catastrophe requiring emergency surgery. The most commonly reported of such complications are obstruction and perforation.

A case is reported in which a nonopaque object (a toothpick) was demonstrated before the necessity for surgical intereference became clinically apparent. The history was not helpful, and the symptoms might have suggested a functional disorder had the roentgen findings been noncontributory.

The mucus-encased toothpick was shown as a tubular filling defect in the third portion of the duodenum at the ligament of Treitz, and caused a "stretching" deformity of the intestinal lumen. Delayed films showed residual barium coating the object.

The author found, in the literature of the past twelve years, no other report of roentgen demonstration of a nonopaque intestinal foreign body prior to the development of major complications; he suggests that such findings could be made more frequently.—*Lynne Taylor, M.D.*

Roberts, Sava M., Peskoe, Louis Y., and Fader, David E. Adenoma of the jejunum; case report. *Radiology*, Sept., 1952, *59*, 409–414.

An adenoma of the jejunum was observed for approximately three years before removal by surgery. During this time, the adenoma increased in size but did not cause obstruction.

The authors believe preoperative diagnosis is possible when a lobulated mass with flecks of barium in depressions is seen, this mass dilating the surrounding and proximal lumen. The dilated lumen may show prominent circular folds giving the appearance of numerous loops of bowel superimposed on one another. Surgery was not undertaken sooner owing to the medical condition of the patient.—*Martin B. Goodwin, M.D.*

Steinbach, Howard L., Lyon, Richards P., Smith, Donald R., and Miller, Earl R. Extraperitoneal pneumography. *Radiology*, Aug., 1952, *59*, 167–176.

The authors describe a technique and procedure for injecting air (oxygen in this series) into the extraperitoneal areolar tissues to demonstrate roentgenologically the posterior abdominal and pelvic viscera. The gas is injected into the areolar tissue between the rectum and sacrum, from which it extends into the cellular tissue of the entire body.

Roentgenograms taken with the patient lying in various positions allow the demonstration of uterus, fallopian tubes, ovaries, kidneys, bladder, adrenals, liver and spleen. It was not possible to outline the pancreas in any of their patients. With larger quantities of gas, mediastinal and neck structures may be outlined.

The procedure apparently is a safe one and no complications have occurred in the patients thus examined.—*Donald N. Dysart, M.D.*

Smith, Grafton A. A study of intestinal intubation using a flexible stylet with controllable tip. *Surgery*, July, 1952, *32*, 17–30.

Whereas parenteral saline was found to prolong the lives of dogs with high intestinal obstruction, it did not have a similar effect in cases of low intestinal obstruction. This difference in reaction has been thought to be due to absorption of toxic substances in cases of low intestinal obstruction. The work of Wangensteen and others is cited regarding the unfavorable effects of intestinal distention in cases of obstruction, and the importance of a combination of electrolyte therapy and decompression is emphasized. In this regimen, the Miller-Abbott tube has played an important part, although it has had certain drawbacks which have stimulated the development of modifications in its structure and use. One such modification which makes use of a specially constructed tube and stylet is described in this article.

The stylet is inserted through a hole in the side of the tube to within 1 inch of the stainless steel endplug. The tube is introduced through the mouth into the stomach, with the patient supine on the fluoroscopy table. The manipulations required to pass the tip of the tube into the jejunum are described. All manipulations are done with the use of fluoroscopy.

An intubation is considered successful when the tube reaches the distal aspect of the second portion of the duodenum. The author states that an average passage time in 162 successful intubations was about eleven minutes. Of 24 patients with gastroenterostomies, 23 were intubated in an average time of seven minutes.

In a series of 180 cases in which intubation was carried out there were no perforations, and the author commends his procedure as being relatively safe.

Of the 180 patients, a group of 113 having intestinal obstruction was carefully studied to determine the effectiveness of intestinal suction. In 83 per cent of the patients with intestinal obstruction decompression was achieved within twenty-four hours.—*André Bruwer, M.B., Ch.B.*

Franklin, Alfred White, and Hosford, John P. Meconium peritonitis due to a hole in the foetal intestinal wall and without obstruction. *Brit. M. J.*, Aug. 2, 1952, *2*, 257–259.

Two cases of meconium peritonitis due to perfora-

tion, but without intestinal obstruction are reported. Both patients survived following operation. One exhibited evidence of pancreatis fibrosis and the other was apparently normal.

The presence of meconium in the fetal peritoneal cavity produces an intense chemical peritonitis. The visceral peritoneum becomes thickened to form a membrane encasing the intestines; in it and in the thickened parietal peritoneum calcium is deposited, recognized roentgenologically and histologically. In some cases there is a plastic peritonitis, in others gross ascites. Such changes are not due to infection, but infection is added when the normal intestinal bacteria appear on the third or fourth day of life.

It is estimated that in only half of the cases was there evidence of intestinal obstruction and a hole may be found without any obstruction. Other cases have been reported in which subsequent postmortem examination failed to show any hole through which meconium could have escaped. The authors suggest that it is possible that a fetus with meconium ileus may by vigorous peristalsis rupture the gut and relieve itself by passing the meconium into the peritoneal cavity, and that the perforation may even heal in some cases before birth.

The clinical picture is that of neonatal small intestinal obstruction with massive abdominal enlargement. Aspiration of the abdominal contents reveals a thick fluid, albuminous, heavily bile stained and containing fat, cholesterol and epithelial squamae on microscopy. The roentgenogram of the abdomen may reveal calcification and gross dilatation of the small intestine. If gas cannot be seen in the rectum in the lateral view at or after twenty-four hours, an obstruction in the intestinal tract is presumed to be present.—*James F. Martin, M.D.*

TEMPEST, MICHAEL N. Meconium peritonitis; report of a case due to spontaneous intrauterine perforation of the transverse colon, with a brief review of the literature. *Brit. J. Surg.*, July, 1952, *40*, 28–31.

Meconium peritonitis is a sterile reaction in the peritoneal cavity of the newborn infant due to the escape of intestinal contents. It may occur during intrauterine life or develop immediately after birth. Approximately 50 per cent of the cases demonstrate positive evidence of organic intestinal obstruction. The lesions vary from congenital intestinal atresia to adhesions, intussusception, and volvulus. The remaining 50 per cent of the cases occur without demonstrable organic obstruction. The cause of these perforations is not clear and may occur in any part of the small or large intestine. In many cases, the perforations may be found in segments of gut which are clinically and microscopically normal. The perforation usually closes before birth; frequently the site of perforation cannot be identified at operation or at autopsy.

The author reports a case of a newborn infant with meconium peritonitis which was secondary to a perforation in the transverse colon. There was no evidence of intestinal atresia, impacted meconium or macroscopic weakness of the gut wall. The whole of the small gut was densely matted together by a greenish-yellow plastic exudate. The transverse portion of the colon was involved in the mass. There was no evidence of overdistention of the intestinal segments, as found in long-standing intestinal obstruction; and no calcified masses were found inside the abdomen.—*James F. Martin, M.D.*

FORSHALL, ISABELLA, HALL, E. G., and RICKHAM, P. P. Meconium peritonitis. *Brit. J. Surg.*, July, 1952, *40*, 31–40.

The authors report 3 cases of meconium peritonitis. In 2, there was evidence of generalized peritonitis and in the third, it was localized. They define meconium peritonitis as one which is primarily caused by, and shows evidence at operation or autopsy of characteristic lesions produced by intraperitoneal meconium. They would exclude peritonitis which has been described in the fetus or newborn infant not associated with a present or past perforation of the gut, and also peritonitis arising from a rupture of the intestine during or after delivery in which the escape of meconium is rapidly followed by bacterial infection of the peritoneum.

In none of the 3 cases was a perforation seen at operation, nor was there clear evidence on microscopy of the exact site of rupture. Intraperitoneal calcification occurs but it may be so slight as to be demonstrated only when the excised tissues are roentgenographed. It was impossible to say with certainty how the lesions encountered were produced. The authors suggest the following possibilities from a study of their cases: (a) volvulus with perforation secondary to interference with the blood supply of the bowel permitting meconium to escape into the peritoneal cavity followed by fibrosis sufficient to obliterate the lumen and leave only a fine impermeable strand; (b) meconium ileus with pressure necrosis of the mucosa followed by perforation of the gut wall and fibrosis with complete obliteration of the lumen. Other authors have suggested: (a) healing of intrauterine ulcerations of the intestine with subsequent atresia; (b) the presence of congenital endarteritis of the mesentery.

In most instances of meconium peritonitis there is an intestinal obstruction due either to meconium ileus, stenosis, atresia, volvulus or hernia.

The autopsy findings in the 3 cases are reported. One patient demonstrated findings typical of cystic fibrosis of the pancreas. In another, the pancreas was described as normal while the findings are not recorded in the third case.—*James F. Martin, M.D.*

MACHELLA, THOMAS E., DWORKEN, HARVEY J., and BIEL, FRUCTUOSO J. Observations on the

splenic flexure syndrome. *Ann. Int. Med.*, Sept., 1952, *37*, 543–552.

The authors describe the findings in a group of 40 patients to which they have applied the term "splenic flexure syndrome." In most of these cases the findings simulated coronary artery disease. They believe that the distention of the splenic flexure by gas or possibly by feces might be responsible for the symptoms. The symptoms have been reproduced by inflation of a balloon in the splenic flexure. Important differential diagnosis is that of coronary artery disease.—*Allison E. Imler, M.D.*

Goin, Lowell S. Some obscure factors in the production of unusual small bowel patterns. *Radiology*, Aug., 1952, *59*, 177–184.

The effects of psychic and emotional factors and the influence of allergens upon the small bowel pattern are described. Two case histories and accompanying roentgenograms showing the normal small bowel pattern along with the unusual pattern produced under emotional stress are presented. The abnormal pattern was characterized by hypomotility, almost complete disappearance of mucosal folds, and segmentation of the small bowel.

Two case histories of patients allergic to milk are also presented. The normal small bowel pattern of the patients is compared with roentgenograms made following the ingestion of milk. The abnormal small bowel again demonstrates a marked alteration in motility, narrowing and segmentation with barium scattered in an irregular and disconnected manner. There was a large gastric retention.

The author concludes his discussion with the thought that there may be many other causes which may alter the behavior of the intestinal tract.—*Everett E. Seedorf, M.D.*

Reussi, Carlos, Rigoli, Mario H., Braga, Roberto, and Olcese, Edgardo. Los tumores del intestino delgado; su diagnóstico radiológico. (Tumors of the small intestine; their roentgen diagnosis.) *Prensa méd. argent.*, Aug. 15, 1952, *39*, 1916–1924.

The roentgen examination is the procedure of greatest diagnostic importance in identifying tumors of the small intestine. A film of the abdomen with the patient in an erect posture should precede studies for small intestinal motility in order to ascertain the presence of classical fluid levels if an obstruction is suspected. The motility of the small intestine is studied fluoroscopically and with serial roentgenograms made using compression. In recent years the rapid procedure described by Weintraub, Williams and Ettinger has been utilized also. In addition to the study of the colon by the ingestion of barium, a retrograde barium enema is used for demonstrating the insufficiency of the ileocecal valve and for evidence of disease in the terminal ileum.

The roentgenologic signs found in tumors of the small intestine are: (1) localized annular or napkin ring constriction; (2) persistent filling defect of an intraluminal tumor; (3) chronic intestinal stenosis with an exaggeration of the mucosal pattern; (4) dilatation of an affected intestinal segment as the result of infiltration of a neoplasm with obliteration of the mucosal pattern and evidence of stiffening of the intestinal wall. These changes are observed more frequently in sarcomas rather than carcinomas; (5) infiltration of a neighboring loop; (6) signs of intussusception. Not infrequently in carcinomas, the only finding will be that of chronic obstruction of the jejunum or ileum without definite localization of the lesion.

The intestinal tumors studied included; (1) an adenocarcinoma of the third portion of the duodedum; (2) adenocarcinoma of the jejunum; (3) undifferentiated carcinoma of the proximal ileum; (4) polypoid adenoma of the terminal ileum; (5) lymphosarcoma of the jejunum; (6) lymphosarcoma of the ileocecal valve; (7) carcinoid of the distal ileum; (8) Hodgkin's disease, primary in the duodenum; (9) Hodgkin's disease, primary in the jejunum.—*Murray M. Friedman, M.D.*

McEwen, Kenneth L., and Lodmell, Elmer A. Acute fulminating idiopathic ulcerative colitis; report of a case showing unusual roentgenologic and clinical features. *Radiology*, Aug., 1952, *59*, 185–189.

An unusual case of acute fulminating ulcerative colitis is presented, which tends to add to the evidence that this condition is one in which the roentgen appearance of the colon is not a reliable prognostic guide; that the proctosigmoidoscopic findings do not always indicate accurately the extent of mucosal damage; and that no definite criteria exist by which a long-range prognosis can be established in any given case.

A twenty-three year old white male, shortly after rather severe emotional trauma, developed progressively distressing gastrointestinal symptoms, including diarrhea which became bloody. Proctosigmoidoscopic and barium enema studies, twelve days after onset of symptoms, were consistent with acute fulminating ulcerative colitis. The latter study showed, in addition to the usual features, a striking picture of multiple, shaggy, undermining ulcerations deeply penetrating the mucosa, being particularly numerous from the rectosigmoid junction to the midpoint of the transverse colon.

Supportive therapy and aureomycin were given; and seventeen days after the initial barium enema study, the extensive mucosal ulcerations had disappeared, leaving only residual irritability, particularly of the sigmoid and descending portions. During and

briefly following this period the patient became asymptomatic.—*Lynne Taylor, M.D.*

Nushan, Harry, and Miller, Benjamin. Ameboma of the transverse colon. *Ann. Int. Med.*, Aug., 1952, *37*, 372–379.

The authors report the unusual location of an ameboma in the transverse colon. In order of frequency the location of this type of lesion is first in the cecum, then in the sigmoid and then in the descending colon.

The granulomatous mass associated with the ameboma follows chronic amebic ulceration. Partial obstruction or acute intussusception and acute occlusion may result.

The diagnosis of ameboma may be difficult because the history may be misleading. The symptoms consist of localized abdominal pain and emaciation. Nausea, vomiting and signs of intestinal obstruction may be present. A palpable tumor is usually present and multiple tumors may be found. The presence of amebae in the stools may be difficult or impossible to demonstrate.

The differential diagnosis is important because the usual therapy of ameboma is primarily medical. Carcinoma of the colon is the lesion which may simulate the ameboma most closely. Differential diagnosis depends upon: (1) carcinoma is usually single while amebomas may be multiple; (2) in amebiasis a larger segment of bowel is involved and the transition from normal to abnormal is much more gradual; (3) in amebiasis the mucosal pattern appears more regular, the bowel wall is not as rigid and the obstruction is not as marked as in carcinoma; (4) following antiamebic therapy the lesion disappears and the bowel returns to normal.

The authors' patient was a fifty-four year old male who complained of loss of weight, anorexia and weakness. Barium enema examination showed a lesion in the transverse colon compatible with an early carcinoma. At laparotomy the transverse colon showed redness and thickening of an area about 8 cm. in length. Resection and end-to-end anastomosis were performed. During hospitalization this patient subsequently developed a gastric ulcer which necessitated a subtotal gastrectomy.—*J. N. Ané, M.D.*

Roman, Paul W., Wagner, John H., and Steinbach, Stanley H. Massive fatal embolism during barium enema study. *Radiology*, Aug., 1952, *59*, 190–192.

In the reported case, a Bardex bag was inflated under fluoroscopic control, and the rectum was filled with barium. The patient began to strain down, and the injected barium was seen to assume a peculiar stringy appearance, conforming in shape to the pelvic vessels adjacent to the rectum. Although the examination was immediately discontinued, the patient died within four or five minutes. Postmortem study showed barium in the inferior vena cava, the hypogastric vein, certain pulmonary vessels, renal vessels, and several glomeruli. The mechanism of embolization could not be ascertained, though possibly the barium entered the venous system through a fissure or a varix.—*Lynne Taylor, M.D.*

Genitourinary System

MacLean, John T. Injuries to the genitourinary tract. *Canad. M.A.J.*, Sept., 1952, *67*, 220–231.

The author comments briefly on the common types of injuries to the genitourinary tract and suggests methods of treatment.

Conservative means are recommended in the management of rupture or trauma to the kidney, until this proves to be an untenable position. Eventual return of function often occurs, but if it does not, nephrectomy may be performed with less risk to the patient later. The author draws attention to the cassette holder designed by Dr. L. Orkin of New York, for use with 14 by 17 inch cassettes and a Lysholm grid. The use of this device is valuable for portable studies of the abdomen in those patients who cannot, because of their injuries, be moved.

Rupture of the bladder occurs intraperitoneally in 40 per cent of cases. Rupture of the bladder, either intra- or extraperitoneal, calls for closure of the tear, suprapubic drainage and soft tissue drainage of the extravasated urine.

Regarding urethral injuries, methods of using a urethral catheter as a splint to produce healing with or without surgical repair of the tear are described. Late repair and graft of the urethra are also discussed.—*Daniel L. Rousseau, M.D.*

Martinez, Adaucto. Calculose ureteral pélvica. (Pelvic ureteral calculosis.) *Rev. med. e cir. de São Paulo*, April, 1952, *12*, 215–250.

Utilizing 30 illustrative cases, the author discusses salient points in the diagnosis and treatment of calculi in the pelvic portion of the ureter.

Roentgenographic examination must be considered the most valuable aid in the diagnosis and may be done with or without contrast media. The great majority of the ureteral calculi are radiopaque and can easily be demonstrated by a plain roentgenogram. Confusing shadows due to phleboliths, calcified lymph nodes or sclerosis of the pelvic vessels must be excluded. Retrograde pyelography is superior to excretory pyelography.

Exact localization of the calculi and determination of the degree of pyeloureteral dilatation are important factors in the choice of therapeutic procedures. —*Ernst A. Schmidt, M.D.*

Higgins, Charles C. Bilateral polycystic

kidney disease. *A.M.A. Arch. Surg.*, Aug., 1952, *65*, 318–329.

The author reviews 94 cases of congenital polycystic kidney disease observed at the Cleveland Clinic from January, 1935, to January, 1951.

Polycystic kidney disease is probably always bilateral, and consists of massive involvement of the renal parenchyma by cysts of various sizes and number. The course is slow and progressive. Various theories have been suggested to explain the congenital factors responsible. Most investigators agree that kidney cysts arise from a failure of proper fusion of the secretory and excretory systems during embryonic development.

Polycystic kidneys frequently become very large and may weigh from 15 to 18 pounds. The kidney surface is irregular and nodular.

Pain and hematuria are the outstanding symptoms. Chills, fever, lassitude, weakness and loss of weight are the symptoms of secondary importance. About 50 per cent of the patients complained of headache and in most of these cases hypertension was present. In the author's series the youngest patient was eighteen years of age and the oldest sixty-two. There were 42 men and 52 women.

Roentgenographic examination is essential in the diagnosis of polycystic kidney disease. Renal enlargement and coexisting lesions, such as calculi and abnormality of the bones, are discovered on the initial roentgenogram. Intravenous urography is indicated if the blood urea is within normal limits. In 65 cases excretory urography was performed. In 33 of these a definite diagnosis was made. In 17 cases tentative diagnoses were established and in 15 cases there was insufficient concentration of the contrast medium for diagnostic purposes. Retrograde pyelography was performed in 56 cases, and correct diagnoses were evident in 51 instances.

Early in the course of the disease roentgenographic diagnosis may be difficult. Later with enlargement and increase in the number of the cysts the calyces become deformed, angulated and elongated. Still later the typical "dragon deformity" may be noted. Of importance is the bilateral presence of the lesions. —*J. N. Ané, M.D.*

Ercole, Ricardo. Radiología clínica del ántrax del riñón. (Clinical radiology of anthrax of the kidney.) *Arch. españ. urol.*, 1952, *8*, 221–244.

The author has observed 14 patients with anthrax involving the kidney. The pyelographic image simulated tumor formation, without signs of infiltration or of invasion of the excretory passage, which may be seen with neoplasm. Perinephric infection may be present. Administration of antibiotics, such as penicillin, during a febrile stage may ameliorate the clinical course. A palpable mass may be present. Roentgenograms and diagrams of the 14 cases are presented.—*Charles M. Nice, M.D.*

Skeletal System

Vines, R. H. Polyostotic fibrous dysplasia. *Arch. Dis. Childhood*, Aug., 1952, *27*, 351–355.

Two boys with polyostotic fibrous dysplasia are described with findings of café au lait spots, cystic changes in expanded bones with thinned cortices and a "ground glass" appearance. One patient sustained a pathological fracture, the other developed bossing of the skull with sclerosed facial and cranial bones. Both had an elevated serum alkaline phosphatase and a family history of skin pigmentation. Because of an incidence of skin pigmentation found to be 12 per cent in the general population the significance of this finding is doubtful. Neither showed precocious puberty.

The etiology and relationship to the monostotic form of the disease are discussed as are the pigmentary, endocrine, and chemical changes.

A review of 140 cases in the literature did not prove an increase in the number of congenital anomalies associated with this disease. Life expectation is not altered. Progression in adult life rarely occurs. Fractures heal well.—*Fay H. Squire, M.D.*

Lichtenstein, Louis, and Hall, John E. Periosteal chondroma; a benign cartilage tumor. *J. Bone & Joint Surg.*, July, 1952, *34-A*, 691–697.

Periosteal chondroma is described in this paper as a slowly growing benign cartilage tumor of limited size which develops within and beneath the periosteal connective tissue and characteristically erodes and induces sclerosis of the contiguous cortical bone. As such, it must be clearly distinguished from osteocartilaginous exostosis (osteochondroma) and from solitary enchondroma. Thus far Lichtenstein and Hall have encountered it on hand and foot bones, as well as on large limb bones, as for example on the tibia and humerus. It seems likely, however, that the lesion also occurs in other sites.

Periosteal chondroma is composed of lobules of hyaline cartilage, not unlike an enchondroma but tends to be more cellular and its content of plump cartilage cell nuclei is likely to suggest a significantly active growth potential, although this is not borne out by what the authors have found to be its clinical behavior. The tumor tends to develop in close apposition to the cortex of the bone, nestling in a semilunar hollow which it has apparently created by gradual pressure erosion.

The lesion may develop in children as well as in adults. The symptoms usually referable to it are pain, gradual swelling and local tenderness. The duration of symptoms in the cases observed ranged

from a few months to ten years. Clinically, a firm, generally small, slightly tender tumor is found which, if located near a joint, may produce some limitation of motion. At operation, a rubbery, firm, lobulated cartilage tumor adherent to the periosteum is found.

On roentgen examination, a comparatively small, peculiar trough-like hollow or declivity in the cortex is noted. The base of this hollow, semilunar in shape, consists of sclerotized cortical bone, apparently reflecting reaction to slow pressure erosion. Of the tumor itself, which is responsible for these changes, beyond a vaguely defined soft tissue shadow, only such focal areas as happen to be sufficiently calcified or ossified for visualization are observed. If the projection is such that the depressed, sclerotized cortical base is superimposed on the shaft of the bone, one may gain a false impression of a central tumor of rounded or ovoid contour suggesting an enchondroma. Ordinarily, the peripheral contour of the tumor is not visible, although in one instance in the humerus the peripheral contour became outlined by a delicate convex shell of periosteal new bone. The authors state that if one is familiar with the roentgenographic appearance of periosteal chondroma, the picture in any given instance is likely to be sufficiently distinctive to strongly suggest the diagnosis, although otherwise it will probably be rather puzzling. This will be heightened if, as is sometimes the case, one observes irregular focal areas of increased density within the lesion nestling in the hollow of the eroded cortex, which indicates its probable cartilaginous nature. The possibility of a glomus tumor or a lesion of pigmented nodular tenosynovitis (giant cell tumor of tendon sheath, so-called) which has eroded the contiguous cortex, must be considered in the differential diagnosis if the tumor occurs in the phalanges of the fingers.

The treatment which is recommended in most instances is conservative surgical extirpation and curettement of the eroded, sclerotized cortical base. Block excision is feasible if the tumor has involved a long limb bone and is substantially larger than it is on a finger phalanx, for example. The results of treatment by either method were uniformly satisfactory. The authors would be disinclined to advocate trial of roentgen therapy in view of the ineffectiveness of irradiation of cartilage tumors in general. Moreover, they feel that it scarcely seems justified to accept the known hazards of roentgen therapy in treating a benign neoplasm which can be readily cured by simple surgical removal.—*Ralph S. Bromer, M.D.*

Nunziata, Américo, and Maslo, Pinkus. "Periostitis albuminosa" de Ollier y Poncet. ("Albuminous periostitis" of Ollier and Poncet.) *Prensa méd. argent.*, July 18, 1952, *39*, 1651–1659.

A twenty-three year old patient presented with a fusiform tumefaction of the distal right thigh. No special historical antecedents were elicited. Pain was less on resting, augmented on ambulation. Diathermy exaggerated the complaint. The femoral diaphysis on roentgenograms revealed a lacunar cortical fissure which elevated and separated the external layer. Periosteal proliferation was noted. On surgical exploration about 50 cc. of clear, seroalbuminous fluid was found in a thinly encapsulated area. Drainage was instituted and postoperative course was uncomplicated. Histopathologic diagnosis was "chronic inflammatory process with intense granuloangiomatosis, abscess formation and presence of necrobiotic zones." It was concluded that this represents a case of chronic osteoperiostitis, similar to the disease of Ollier and Poncet.

Relatively few examples of this disease are found in the world literature. Moulonguet and Rousset considered it a forme fruste of osteomyelitis. Like osteomyelitis, the condition is seen in children and adolescents. Also, it occurs near the epiphyses. The staphylococcus has often been indicted as the etiologic agent. The condition has been associated with fractures.

The authors conclude that the type of fluid and tissue changes do not indicate that this is a forme fruste of osteomyelitis, but rather a distinct clinical condition, readily cured by surgery.—*Charles Nice, M.D.*

Dennison, Wallace M., and Macpherson, Duncan A. Haematogenous osteitis of infancy. *Arch. Dis. Childhood*, Aug., 1952, *27*, 375–381.

Osteitis of infancy differs from osteitis in children over two years of age. Two groups are recognized: (1) streptococcal osteitis of infancy, in which toxicity is uncommon and although joint infection and abscess formation are frequent, sequestration is rare and bone defects are restored to normal as are joint functions; (2) osteitis of the newborn, which may be either benign or severe. The organism is almost invariably a coagulase positive staphylococcus, often resistant to penicillin but sensitive to aureomycin and streptomycin.

The benign form of osteitis of the newborn involves one bone with little systemic disturbance and the prognosis is good even without antibiotics. The severe type, known also as generalized osteitis of the newborn, is a grave systemic disease with multiple swellings, positive blood cultures, and roentgenographic findings of subperiosteal bone formation and destruction of the metaphyses and epiphyses. Joint infection occurs early and irreversible bone changes are noted. Seven cases of this type are described.

Treatment of these diseases depends on the sensitivity of the organism to the various antibiotics. Evacuation of pus and aspiration of joints should be

performed as in older children. The progress of the bone disease in the severe type is influenced little by antibiotics and the prognosis as to function is guarded.—*Fay H. Squire, M.D.*

Giaccai, L. Familial and sporadic neurogenic acro-osteolysis. *Acta radiol.*, July, 1952, *38*, 17–29.

Familial Neurogenic Acro-osteolysis.

This condition is characterized by chronic ulceration resulting in extensive mutilation of the feet, sensory disturbances of the extremities and a familial incidence.

1. Symptoms and signs. More than one successive generation may be involved. Males are affected three times as frequently as females. The disease usually begins in childhood. A painful ulcer appears on the plantar aspect of the foot and after some time, a bony spicule is discharged and the ulcer heals. After an interval another swelling may appear and another region on the sole of the foot is involved. The feet become deformed, chronically swollen and extensively mutilated.

Sensory disturbances are always associated with the foot lesions. Superficial sensibility is impaired, the sensation of pain being the one most affected.

2. Roentgen appearance. The bone changes characteristic of familial acro-osteolysis are usually complicated by a superimposed chronic osteomyelitis involving the bone at the site of the ulceration. The heads of the first and second metatarsals are usually the first to be involved by the osteomyelitic process.

Changes typical of familial acro-osteolysis are most evident in the phalanges. The shafts are thinned out and the epiphyses remain unaffected until the later stages. When the shafts have been completely destroyed, the epiphyses remain as loose bone fragments which either undergo slow reabsorption or are eliminated as sequestra.

Sporadic Neurogenic Acro-osteolysis.

As well as being non-familial, sporadic acro-osteolysis differs from the familial type in the following ways:

1. It is often unilateral.

2. The sensory disturbances of the foot are inconstant and if present, are usually not very conspicuous.

3. This type is almost always associated with demonstrable malformations of the spine and of the foot.

4. There is no definite sex incidence.

5. The progress of the disease is more rapid; within a few years the maximum degree of mutilation is reached and the condition then becomes stationary.

In other respects the clinical features of sporadic acro-osteolysis do not differ from those of the familial type. Recurring ulcers and elimination of sequestra mark the progress of the disease until extensive mutilation of the foot results.

Etiology. The author believes that both types of acro-osteolysis are due to a peripheral nerve lesion but the type of nerve involvement is obscure. The following facts have caused him to arrive at this conclusion:

1. The osseous lesions are most marked at the distal ends of the feet.

2. The sensory disturbances have a peripheral distribution.

3. The type and distribution of the osseous lesions in acro-osteolysis bear a close resemblance to the changes characteristic of neural leprosy.

4. The normal appearance of the spinal cord at the autopsy of one of the author's cases suggested that the lesion in the sensory paths may have been located in the peripheral fibers.—*Mary Frances Vastine, M.D.*

Plenk, Henry P. Calcifying tendinitis of the shoulder; a critical study of the value of x-ray therapy. *Radiology*, Sept., 1952, *59*, 384–389.

Calcifying tendinitis of the shoulder was observed most commonly in the middle-aged group of people and, in this controlled study of the effect of roentgen therapy on 38 patients, was seen more frequently in females. Films of the shoulder in external and internal rotation of the humerus demonstrated calcification most commonly in the supraspinatus tendon with a smaller number showing it in the tendon of the infraspinatus muscle. None was found in the teres minor or subscapularis tendons. Calcium deposition in the subdeltoid bursa, seen best on the external rotation film as a thin layer of increased density extending 2 to 3 cm. below the greater tuberosity, was noted in several patients. This bursal calcification was thought to occur when the tendon ruptured and discharged the calcareous material into the bursa.

The author discusses the life history of this disease and presents the results of his treatment. Seventeen of the 38 patients actually received no irradiation as a 5 mm. lead shutter was left in place during the treatment without the patient's knowledge. The evaluation carried on for a period of six weeks after the treatment indicated that equally satisfactory results were obtained in both the treated and untreated groups. Since an average of eleven days was required for definite improvement or relief of symptoms in both groups it was felt that roentgen therapy could not be credited with shortening the period of disability. Complete restoration of motion of the shoulder was evident at the end of six weeks in an equal number of the treated and untreated cases.—*Donald N. Dysart, M.D.*

Wieland, H. Ein Beitrag zur Kenntnis der Acroosteolyse. (A contribution to the knowl-

edge of acro-osteolysis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Aug., 1952, *77*, 193–198.

The author reports the case history of a patient with osteolytic lesions in the phalanges of both hands and feet. This case is similar to the one described by Harnasch (*Fortschr.*, 1950, *72*, 352). The latter author believes that the lesions are caused by a central nervous disturbance and inadequate function of the anterior lobe of the pituitary. Reproduced roentgenograms show large osteolytic defects and occasionally there is complete absence of the distal phalanges of the toes and fingers. The roentgen appearance strongly resembles that seen in neuropathic or neurovascular disturbances (such as, for instance, leprosy).

The author also mentions that the patient was suffering from psoriasis.—*Egon G. Wissing, M.D.*

Junge, H. Über diagnostische Befunde bei Peridurographie mit Ausnahme hinterer Bandscheibenvorfälle. (Diagnostic findings in peridurography with exception of cases of posterior prolapse of the intervertebral disc.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Aug., 1952, *77*, 187–192.

This method consists of a peridural injection of viscous perabrodil M (plus 1 per cent novocaine) into the sacral area.

The author describes the use of this method in displacement of vertebrae, inflammatory disease of the spine, spinal injuries and ankylosing arthritis.

The conclusion is reached that the method cannot be recommended in as much as severe accidents may occur if this opaque medium is injected intradurally. —*Egon G. Wissing, M.D.*

Morton, Daniel G., and Gordan, Gilbert. Observations upon the role of the sex hormones in the development of bony pelvic conformation. *Am. J. Obst. & Gynec.*, Aug., 1952, *64*, 292–300.

The authors believe that there is a remarkable similarity between the prepuberal pelves of children of opposite sexes, and that if the differences occurring with maturation were due to sex hormones, then a study of the pelves of hypogonadal individuals should show little or no differentiation according to sex. However, they found that there were definite sexual distinctions, even in the presence of very low hormonal levels.

They conclude that the pelvic characteristics of mature pelves do not depend primarily on sex hormones, but that they may have some effect on maturation.

They suggest that roentgenographic study of the pelvis may be of aid in studying hypogonadal individuals in the absence of secondary sex characteristics.—*George L. Sackett, Jr., M.D.*

Snodgrass, Thomas J., and Suckle, Henry M. Sacrococcygeal chordoma. *A.M.A. Arch. Surg.*, July, 1952, *65*, 181–187.

A total of 144 cases have been recorded in the literature including the case reported by the authors who have also reviewed 8 additional cases between 1943 and 1948.

Chordomas may occur at any age but are most frequent in the fifth decade of life.

It is a tumor which arises from the primitive notochord, progresses slowly, tends to invade and destroy bone by direct extension and occasionally metastasizes to distant lymph nodes or viscera.

The tumor becomes invasive only when it meets with some resistance to its growth as the sacrum. Rarely, widespread metastases do occur in the lungs, liver and peripheral lymph nodes. Radiation therapy has very little effect upon the growth and the only effective treatment is surgical excision. It is frequently necessary to re-operate several times for local recurrence.

The authors' case of chordoma of the notochord is reported in detail.—*W. W. Maver, M.D.*

Kite, J. H., and French, G. O. The early diagnosis of flat-headed femur. *South. M. J.*, July, 1952, *45*, 581–585.

The authors stress the necessity of recognizing the early symptoms and roentgenographic signs so that treatment can be started before much damage has been done to the softened femoral head. The bad end results are due, not so much to the form of treatment, as to the delay in diagnosis which has allowed the head of the femur to become flattened and partially deformed before the diagnosis has been made.

The most common complaint of the 165 cases reviewed was pain in the thigh or knee after an injury or overexertion. At almost the same time a limp was noticed. The first physical findings were limitation of internal rotation and slight limitation of full abduction.

The earliest roentgenographic sign has been reported by Waldenstrom as a slight lateral separation of the head of the femur from the medial wall of the acetabulum. This was present in 99 per cent of the cases. A full description of the sign is quoted.

The next most helpful early sign is the so-called "obturator sign." The obturator foramen is smaller on the diseased side. Although present in a large percentage, the authors feel that it is due to a slight rotation of the pelvis rather than the disease itself, as they have found it in many other conditions.

Another early sign can be called the "roof sign" which has not been described previously. In over a

third of the cases, the lateral half of the head loses its normal convexity and becomes flattened. This produces a head shaped like the roof of a house. The mechanism for this is that the softening of the head and destruction begin first at the lateral and anterior surface.

The fourth early sign is a certain degree of bone atrophy on the involved side which is measured by a photoelectric light meter. A slight change in the early case can be detected by this method when it was not apparent to the eye.—*Norman Heilbrun, M.D.*

Maatz, Richard, and Lempert, Helmuth. Zur Kopfnekrose nach medialer Schenkelhalsfraktur. (Necrosis of the head following medial fracture of the femoral neck.) *Chirurg*, July, 1952, *23*, 304–306.

The authors compare the results obtained in the treatment of 386 cases of femoral neck fractures. The material, which was accumulated from the records of the Schleswig Holstein Compensation Insurance Company, covers the years 1930 to 1949.

They feel that the practice of pinning a hip in an impacted fracture "to give more support" is not only unnecessary but harmful and may produce necrosis of the head by separating the fragments and severing existing blood supply even if only temporarily.

Two hundred and twenty-eight cases were treated conservatively and 158 with the Smith-Petersen pin. Numerous cases of impacted fracture which healed without necrosis after conservative treatment were omitted from this study. The remaining 312 cases were observed for a period of from 3 to 20 years.

In the group treated conservatively, pseudoarthrosis was found in 41 per cent and necrosis of the head in 7 per cent of the cases, while the operated group exhibited pseudoarthrosis in 20 per cent and necrosis of the head in 21 per cent of the cases. In discussing the reasons leading to the necrosis, the authors mention the possibility of injury to the nourishing vessels and the possible action of the nail in the sense of metalosis. They stress especially the fact that the pinning produces immobilization of the fragments and, through the subsequent weight bearing, the head undergoes molding in the process of bone substitution in the presence of a partial or total necrosis. The resulting deformity is then detectable on roentgenographic examination. In the cases treated conservatively, although the necrosis may exist, weight bearing is avoided and consequently there is no deformity of the head.

In conclusion, the authors state that if we accept the fact that good bone union is dependent on correct apposition of the fractured surfaces, the maintenance of such conditions is important so as to avoid necrosis of the head.—*Salomon Silvera, M.D.*

Jenny, Fritz. Über aseptische Nekrosen im Sprungbein. (Aseptic necrosis of the talus.) *Chirurg*, July, 1952, *23*, 300–304.

The author describes the different forms of avascular necrosis which may occur in the talus. In fractures, although all blood vessels in the region of the neck may be severed, a necrosis does not develop unless there is in addition a luxation of the talus body, and the blood supply of the joint capsule and ligaments is interrupted. The roentgenographic examination, after several weeks, reveals normal density of the necrotic fragment, while the adjacent bones show atrophy.

The process of bone substitution takes months and occasionally years. The early immobilization of the fragments is of great importance for the revascularization. Klaiger recommends, in addition, arthrodesis of the inferior joint to promote vascularization from the calcaneus. The author warns against too early weight bearing which may break down newly built bone trabeculae, resulting in an uneven articulating surface at the superior aspect of the trochlea and the production of "arthronosis deformans." Two such cases are presented showing disability and painful symptoms.

Another form of necrosis of the talus is osteochondritis dissecans. The author agrees with Axhausen in recognizing the disease as a primary aseptic bone necrosis, the first pathological change consisting of epiphyseal necrosis followed by subsequent subchondral fracture. He states that if the theory of repeated trauma with disturbance of the blood supply and subsequent necrosis is accepted as the basis of the causation of osteochondritis dissecans, the existing medical judgment of traumatic medicine must be fundamentally revised. Osteochondritis dissecans appears most frequently at the superior and medial aspects of the trochlea. Roentgenographically there is a subchondral process of demarcation of about 1 cm. in diameter and mostly 3 to 5 mm. in depth. A thin zone of increased bone density separates it from the normal bone structure. Eight such cases are presented in 2 of which the loose fragment was found to be surrounded by cartilage at operation.

In concluding, the author describes a case of aseptic necrosis involving the head of the talus and the navicular bone which proved to be of tuberculous origin.—*Salomon Silvera, M.D.*

Uehlinger, E. Die skelettveränderungen bei Leukämie. (Skeletal changes in leukemia.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Sept., 1952, *77*, 263–276.

The relationship between bone marrow and bone tissue is based on the following observations:

1. Heterotopic bone formation is always associated with simultaneous differentiation of the bone marrow.

2. Formation of mature marrow is associated with

differentiation of mature bone. This is particularly striking in osteopetrosis, where the narrow marrow space contains undifferentiated, spindle cell stem tissue without blood cell formation.

3. Bone changes vary, depending upon the age of the displacement of the diseased marrow tissue. With the replacement of the hyperplastic marrow by fatty marrow, the bony structure becomes more normal.

There are many examples of lack of association between the process in the bone marrow and that in the bone tissues. In leukemia, there is lymphatic or myeloid hyperplasia usually without any structural bone changes. Bone changes in leukemia are rare, and are limited to absorption or production of bone. The processes occur not through the blood cells but through osteoblasts and osteoclasts. Absorption is much more frequent than is production. Generalized demineralization usually is present, although occasionally there are localized areas of bone absorption. When bone production does occur, the endosteal and periosteal changes may be differentiated easily. The periosteal thickening is nonspecific and results from a raising of the periosteum by leukemic infiltration. Periostitis is an expression of extra-osseous extension of the myeloid tissues and of exhaustion of the endosteal reserve space. On the contrary, the rare endosteal hyperostosis or spongiosclerosis is a true specific change and is related to osteomyelosclerosis. Histologically, the new bone formation does not arise from that bone in immediate contact with the lymphatic tissue but from an intervening fibrous layer.

The characteristics of the bone changes in leukemia are due to:

1. Leukemia destroys existing tissues.

2. The growth potential of the leukemic tissue exceeds considerably that of the other body tissues.

3. Leukemic tissue process runs in phases, remissions and exacerbations. The bone process may become normal at a favorable stage of the disease.

4. Leukemic marrow process involves the entire skeleton, particularly the peripheral short and long bones.

Leukemic changes in the bones occur much more frequently in children than in adults. No distinction can be made between lymphatic and myelogenous leukemia. In adults, there is diffuse osteoporosis, spotty bone absorption, and localized bone destruction. The diffuse osteoporosis cannot be differentiated from senile changes, on roentgen film alone, except for the more rapid onset in leukemia. The spotty areas of bone absorption are small in size, and round or oval in shape. They simulate myeloma changes, although in the latter, these are more sharply outlined. Localized bone absorption, particularly in lymphatic leukemia, may lead to spontaneous fracture. The roentgen appearance is that of osteolytic metastasis but healing is much more unfavorable than in carcinoma metastasis.

In children, the early bone changes lead to disturbance in the primary and secondary ossification processes, without simultaneously delaying the growth in length or width of the bones. The following single changes are not characteristic but in combination at least suggest the diagnosis: Juxta-epiphyseal band-like osteoporosis, generalized osteoporosis, spotty bone absorption, endosteal osteosclerosis, and periosteal bone production. Juxta-epiphyseal osteoporosis can be made out best in distal and proximal epiphyses of tibia, distal of femur, distal and proximal of radius, and distal of ulna. Anatomically, there is a leukemic infiltration of the epiphyses. In remissions, the juxta-epiphyseal osteoporosis may disappear in several weeks; Landholt claims that it takes at least six weeks for this change to take place.

The moth-eaten spotty areas of osteoporosis in the epiphyses and metaphyses of the long bones and of the short and flat bones are more characteristic of leukemia. Often, there is also cortical erosion. The combination of juxta-epiphyseal band-like osteoporosis, spotty bone absorption, and atrophy of the spongiosa gives a characteristic roentgen appearance, which may be considered pathognomonic for leukemia. The skull may show granular atrophy and erosion of the external table. When periostitis of the long bones is noted, there is always a clear zone between it and the cortex. Endosteal osteosclerosis seldom is seen in children with leukemia. Rheumatoid joint symptoms and findings are the prominent clinical manifestations and mask the hematological disease. Usually there is fever, pain and swelling of the large joints, and more seldom of the fingers and toes. Lymph node, spleen and liver enlargement is not particularly noticeable. The white blood cells usually are not increased but leukoblasts are present. Average course is six months but very few patients survive over two years.

The author states that he has never seen localized or generalized osteosclerosis in leukemia of the adult. Metastatic carcinoma with involvement of the spleen and bone marrow is most often the disease which will give the roentgenological and hematological pictures simulating leukemia.—*Samuel Richman, M.D.*

STODTMEISTER, R., and SANDKÜHLER, ST. Grundsätzliches zur röntgenologischen Beurteilung der Osteomyelosklerose. (Fundamentals in roentgen evaluation of osteomyelosclerosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Sept., 1952, *77*, 283–287.

Fully developed osteomyelosclerosis is characterized by the following triad:

1. Endosteal sclerosis of the spongiosa, with considerable increase of the bone substance, narrowing of the medullary space, and obliteration of the bone marrow by fibrous tissue.

2. Giant spleen, with extra-osseous myelopoiesis.

3. Bone marrow insufficiency, with anemia,

granulocytopenia, thrombocytopenia, and sometimes with a blood picture resembling that of myeloid leukemia.

On roentgen examination, the bones are thickened because of the increase of bone substance. In general, those parts of the skeleton which normally contain cell-forming marrow, are more severely involved. Typically, the thickening is irregular and spotty, although it may be massively homogeneous. The contour of the involved bone is normal.

Bone changes and clinical picture do not run parallel. Extensive osteosclerosis with relatively normal blood findings, and slight sclerosis, with complete breakdown of the blood forming cells, may be seen. Myelofibrosis is not the end-result of osteosclerosis but both processes occur simultaneously. Fibrosis of the bone marrow leads to changes of the vascular apparatus of the marrow. The cause of the changes in the marrow is unknown.

The large spleen and leukemic-like blood changes have been explained as a neoplastic hemoblastosis. Radiation therapy or splenectomy always has resulted in poor results. Because the spleen assumes myeloid metaplasia as a result of fibrosis of the bone marrow, no treatment to impair the function of the spleen should be attempted.

The compensatory myelopoietic tissue of the spleen is extremely radiosensitive and may be influenced even by roentgen examination of the abdomen; therefore, such examinations should be avoided or held to a minimum. The relation between osteomyelosclerosis and polycythemia is of interest. Dameshek believes that every patient with polycythemia vera, if he lives long enough, will end up with obliteration of the bone marrow by fibrous tissue and thus with osteomyelosclerosis. In addition to the characteristic form of osteomyelosclerosis with extra-osseous myelopoiesis (Heuck), there are more rare forms in which there is no extra-osseous myelopoiesis and no splenomegaly. The prognosis in these latter cases is much worse, the patients living a few months or years. In the Heuck form, it is not unusual for a patient to live up to twenty years.—*Samuel Richman, M.D.*

Stark, Jesse D., Adler, Norman N., and Robinson, William H. Hereditary multiple exostoses. *Radiology*, Aug., 1952, *59*, 212–215.

Three cases of hereditary multiple exostoses are reported. A mother and 2 of her 4 children, aged two and six years, were affected. A brief review of the literature is given.—*Martin B. Goodwin, M.D.*

MacEwen, K. F., and Hall, M. R. Osteoid osteoma. *J. Canad. A. Radiologists*, Sept., 1952, *3*, 48–53.

The authors give a brief review of the literature concerning osteoid osteoma, including a table to show the age incidence and location of the 158 cases reported in the literature up to the present time. The most frequent age of occurrence is between eleven and twenty years, with males predominating two to one. The bones of the lower extremity are most frequently involved.

The clinical picture, pathology, etiology and roentgenological appearance are discussed. Eleven additional cases are reported in detail.—*V. L. Peterson, M.D.*

Stenger, A. Über Knochenveränderungen bei im Wachstumsalter entstandenen arteriovenösen Aneurysmen. (Bone changes in the presence of arteriovenous aneurysms developing during the period of growth.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Sept., 1952, *77*, 308–311.

The author reports the clinical and roentgenologic findings in an apprentice, aged fifteen, who had complained of a sensation of heat in the right hand since his sixth year, followed gradually by an increase in the size of the right arm.

Roentgenograms showed osteolytic changes confined to the involved arm, with worm-shaped areas of rarefaction and trabeculations of increased density. The diagnosis of arteriovenous aneurysm of the right forearm was made on clinical grounds and was confirmed surgically. The arteriogram proved to be of little value. Re-examination of another patient who presented a similar clinical syndrome showed similar bone changes.

The author emphasizes that the bone changes are confined to the parts distal to the aneurysm, extending throughout those parts and not being limited to the region adjacent to the aneurysm; they also state that these changes develop only when the vascular abnormality occurs during the period of growth. They believe that the bone alterations are the result of increased collateral vascularization.

The roentgenograms of the 2 cases are reproduced. —*Albert Oppenheimer, M.D.*

Blood and Lymph System

Schultz, Alvin L., and Hall, Wendell H. Clinical observations in 100 cases of infectious mononucleosis and the results of treatment with penicillin and aureomycin. *Ann. Int. Med.*, June, 1952, *36*, 1498–1512.

The authors deal with the various manifestations of infectious mononucleosis. It was found that about the only constant factors in these cases were: (1) atypical lymphocytosis, (2) abnormal liver function tests, (3) clinical findings, particularly fever and lymphadenopathy, (4) a heterophil antibody titer

of 1:56 or greater. Other protean manifestations of the disease are discussed and several complications are also considered.

In this series 36 patients were treated with penicillin and 15 with aureomycin. Although about 50 per cent promptly improved, comparison with an untreated control group indicated no reduction in the average duration of symptoms or fever as a result of this therapy.—*William A. Klauber, Jr., M.D.*

SCHWARTZ, A., and FRAENKEL, M. Diversion of venous blood flow through transverse sinuses in one-sided innominate vein obstruction. *Radiology*, May, 1952, *58*, 728–730.

In lesions of the superior mediastinum which cause obstruction of the right innominate vein, the blood returning from the right arm, right shoulder, and the right half of the neck must reach the heart through other channels.

The authors present a case and reproduce several roentgenograms taken during phlebography with diodrast which prove that the returning blood reaches the heart by being diverted up the right internal jugular vein, through the dural sinuses of the skull to the left internal jugular vein, reaching finally the superior vena cava. The roentgenogram of the skull obtained after injection of the contrast medium shows filling of both transverse sinuses and part of the superior sagittal sinus.

The authors believe that this is the first such demonstration of these sinuses by injection into the cubital vein.—*Donald N. Dysart, M.D.*

HASTINGS-JAMES, R. Radiological appearances following haemoptysis. *J. Faculty of Radiologists*, July, 1952, *4*, 44–53.

The present article is not concerned with the lesions which cause hemoptysis but with the roentgen appearances resulting from actual bleeding into the respiratory organs. Since opacities seen on a film may be a mixture of shadows due to blood and shadows due to disease, the problem arises of differentiating the constituent parts.

Various manifestations have been seen after bleeding into the respiratory organs, where hemorrhage has been free, perhaps a half cupful or more of blood. Probably no roentgen changes are apparent after small hemoptyses.

1. *Opacities due to blood in the lung fields.* In some cases the affected area shows a general hazy clouding; in others, mottled opacities may be seen, varying from a few small, irregularly rounded densities to large clumped lesions, looking like masses of cotton-wool in the lung field. The extent of the shadowing will vary with the amount and rapidity of bleeding, the site of origin, and the effect of coughing. The latter may expel the blood or it may result in pumping or aspirating it into other parts of the lung.

The site and character of the lesions are dependent upon the relation of the bleeding point to the bronchial tree (these lesions are often segmental) and the force of gravity, which predominates when the bleeding overflows into the main bronchi.

The opacities due to blood clear quickly. The shadowing is usually maximal at the time of the first film examination. Some clearing is evident in two days, and the shadow frequently disappears within a week. Shadowing which has not changed or which has increased eight days after bleeding is unlikely to be due to blood.

Five cases are cited with illustrative roentgenograms. These show the lesions due to the blood in a pure form. The shadows occurred simultaneously with the bleeding and subsided rapidly after its cessation. The patients were not suffering from diseases likely to give confusing shadows.

2. *Spread of disease seen after hemoptysis.* Tuberculosis is the only disease in which hemorrhage has been claimed to be an important means of spread to fresh areas of the lung. Two cases are cited in which the bleeding coincided with increased activity and extent of the disease. It is frequently difficult to determine whether the spread was caused by the bleeding or whether the activity of the disease resulted in hemorrhage.

3. *Segmental or lobar collapse after hemoptysis.* Instead of flooding the area of distribution of a bronchus, hemorrhage may block it with consequent atelectasis of the parts beyond. An illustrative case is recorded in which expectoration of a clot of blood which was a perfect cast of the bronchus was followed by clearing of the lung.

4. *Appearance of fluid levels.* In some cases a previously empty air-filled cavity or cyst may develop a fluid level following hemoptysis, as a result of hemorrhage into it.

5. *Tumor formation.* Hemorrhage into or around a lesion may give rise to an apparent increase in its size. Such an occurrence, followed shortly by shrinkage to the original size of the lesion, would give one a clue to the proper diagnosis.

6. *Hemopneumothorax.* Hemorrhage may be caused by, or may cause, rupture of a subpleural tuberculous focus, with hemoptysis and hemopneumothorax occurring simultaneously.

7. *Chronic effects.* Permanent structural changes in the lung may result if there are recurrent hemorrhages over a long period of time. Such changes include chronic interstitial fibrosis, brown induration and hemosiderosis. An illustrative case due to mitral stenosis, confirmed at postmortem examination, is recorded.—*Samuel G. Henderson, M.D.*

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SIEMENS

RADIOLOGICAL EQUIPMENT NOW AVAILABLE IN U. S. THROUGH WESTINGHOUSE

The Siemens-Reiniger line of specialized radiological equipment which will include body section, convergent beam therapy, Chaoul therapy, and other units is now being distributed nationally by the X-ray Division of Westinghouse Electric Corporation.

The line of specialized equipment made by Siemens-Reiniger-Werke of Germany includes:

- Universal and transversal planigraphs, angiography device and multiple-leaf diaphragm.
- Convergent beam therapy, pendulum therapy, rotational therapy, all giving high depth dose with low skin exposure without multiple port techniques.
- Chaoul therapy unit, for skin contact treatment.

Further details about the Siemens-Reiniger equipment are available — for example:

The convergent beam therapy unit achieves percentage depth doses of 200 to 300% at a tumor depth of from 4 to 10 cm. In operation, the tubehead describes a spiral path on a spherical sector. This results in a conical beam which can be directed so that the tumor lies in the apex of the beam. The maximum dose is, therefore, concentrated on the tumor area; in areas other than the tumor, the dose diminishes rapidly due to divergence of the rays.

The Siemens-Reiniger Universal Planigraph takes body-section radiographs at 56″ target-film distances with the patient in any position: vertical, horizontal, oblique, or up to 90° Trendelenberg. Foreign bodies or cavities are located with speed and precision by fluoroscopy before radiographing; patient dosage, time, and film usage are reduced to as little as 1/10th that in conventional body-section techniques.

These brief examples indicate the scope, precision, and the unique features of the Siemens line of equipment now handled by Westinghouse. For full details, call your Westinghouse X-ray Representative, or write to Department A-87, Westinghouse Electric Corporation, X-ray Division, 2519 Wilkens Avenue, Baltimore 3, Maryland.

(This equipment will be on exhibit at the Seventh International Congress of Radiology in Copenhagen.)

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Acceptance of the Fluorex fluoroscopic image amplifier has exceeded our expectations, and production schedules are being increased to meet the demand.

The following comments by radiologists indicate the high degree of acceptance the Westinghouse Fluorex is receiving as a practical clinical device.

"I would rather have this (Fluorex) image for diagnostic use than 100 films."

"I find one M. A. at 85 K. V. sufficient radiation for G. I. studies."

". . . the greatest advance in X-ray diagnosis since the discovery of X-ray itself."

"You and your associates and also Westinghouse are to be congratulated on this great development. You are undoubtedly far ahead of everybody in this field and have perfected equipment that is certainly ready to enter the clinical field."

"It is a magnificent job and is going to start us on a new path . . ."

For complete information on performance, delivery, or other matters, ask your Westinghouse X-ray Representative, or write to the address below, Department A-88.

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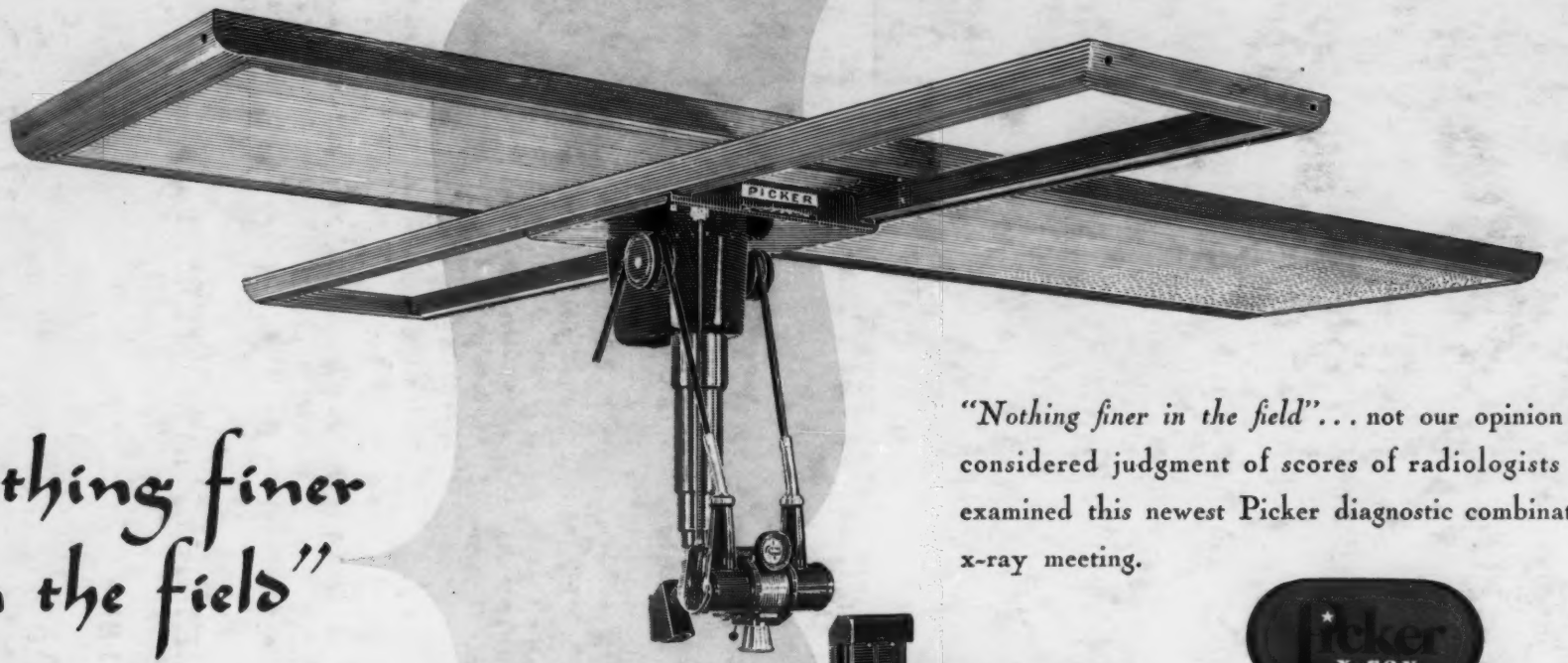

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